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Are heavy drinkers more impulsive than light drinkers?

A comprehensive multi-dimensional assessment of impulsivity in non-dependent heavy drinking young adults

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Awarding institution:
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DCLINPSY THESIS VOLUME II

Service-Related Project and Clinical Case Studies

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Thesis submitted in partial fulfilment for the degree of Doctorate
in Clinical Psychology (DClinPsy)

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SERVICE-RELATED PROJECT

EXPLORING KNOWLEDGE AND AWARENESS OF
COGNITIVE IMPAIRMENTS IN SCHIZOPHRENIA IN A
COMMUNITY MENTAL HEALTH TEAM FOR PSYCHOSIS
A MIXED-METHODS STUDY

SUPERVISED BY DR. ABIGAIL CLARK

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I. INTRODUCTION

I.I) CONTEXT AND OVERVIEW

Cognitive impairment is a fundamental aspect of schizophrenia [1]. The last twenty years or so have witnessed a large, and increasing, body of research literature reporting substantial decrements in cognition in those with the disorder [2]. These problems have serious implications for individuals' functioning [3] and quality of life – and therefore recovery. However, neither the Diagnostic and Statistical Manual for Mental Disorders, Fifth Edition [DSM-V; 4], nor the International Statistical Classification of Diseases and Related Health Problems, Tenth Edition [ICD-10; 5], specify the presence of cognitive impairments as being necessary in their respective criteria for a diagnosis of schizophrenia. Also, although cognitive remediation therapy (CRT) has been convincingly shown to be effective in addressing cognitive impairment [6], the latest NICE (National Institute for Health and Care Excellence) guidelines for psychosis [7] do not advocate its provision for these issues in schizophrenia. For these reasons, despite their being a core feature of the disorder, the extent to which cognitive impairments are currently recognised and addressed by mental health services for those with schizophrenia is at present unclear. This study therefore set out to explore, within an NHS community mental health team for psychosis in the UK, multi-disciplinary staff members' knowledge and awareness of cognitive impairments in schizophrenia.

I.II) LITERATURE REVIEW

The term 'schizophrenia' refers to a heterogeneous range of symptoms, including implausible and peculiar beliefs and sensory experiences, social withdrawal, restricted or inappropriate emotional expression and disorganised behaviour [4]. 'Cognition' incorporates a range of abilities, including: (i) attention/concentration (the ability to focus upon a particular stimulus and to maintain that stimulus in mind, sometimes over an extended period); (ii) short- and long-term forms of memory; (iii) working memory (the ability to briefly hold and manipulate information, e.g., when performing mental arithmetic); (iv) processing speed (the ability to perform simple mental tasks quickly and efficiently); (v) speech and language abilities (e.g., vocabulary, verbal comprehension and verbal expression, etc.); (vi) visuo-spatial skills (the ability to recognise, organise and interpret visual information, e.g., map-reading), and; (vii)

motor skills. An individual's general intelligence (or Intelligence Quotient; IQ) refers to their average ability across these various domains. In addition, executive functions are another crucial aspect of cognition, and refer to a range of processes involved in the initiation and maintenance of goal-directed behaviour [8], including planning/organisation, decision-making, problem-solving, impulse-control, self-monitoring and set-shifting (the ability to 'flit back and forth' between different tasks). Finally, social cognition refers to 'the ability to perceive, process and utilise social information' [9, p. 126] and incorporates theory-of-mind skills (the ability to infer the intentions and mental states of others). Cognitive impairment, therefore, refers to dysfunction in one or more of these abilities.

Historically, the pioneering German psychiatrists Emil Kraepelin [10, 11] and Eugen Bleuler [12, 13] held that cognitive impairments were 'fundamental' in schizophrenia and that positive symptoms were merely 'accessory'. Indeed, the very name that Kraepelin gave to the disorder, 'Dementia Praecox', broadly translates as 'premature cognitive decline'. Yet the decades after witnessed a decline in the conceptual importance placed on cognitive impairments, manifested perhaps most clearly in the DSM-V [4] and ICD-10 [5] criteria for schizophrenia, which focus almost exclusively on positive and negative symptoms, without any explicit reference to cognitive deficits [14]. This relative neglect of cognitive impairments may be due, firstly, to positive symptoms being arguably more salient than cognitive deficits, which, by comparison, may be relatively subtle. It may also be attributable to the absence, until around the mid-1950s, of standardised measures for assessing cognition [2]. However, the development of standardised, norm-referenced neuropsychological measures to systematically assess the various domains of cognition [such as the Wechsler Adult Intelligence Scale - Fourth Edition, or WAIS-IV; 15] proved to be hugely important. The subsequent application of such measures to explore functioning in those with schizophrenia, relative to healthy controls, initiated by Shakow [16, 17], has resulted in a large body of research literature reporting substantial cognitive deficits in schizophrenia.

Over the last twenty years or so, a series of meta-analyses comparing those with schizophrenia against healthy controls has provided overwhelming evidence for the existence of substantial cognitive impairments in patients [18-23]. The very largest cognitive impairments (i.e. of around 1.5 pooled standard deviations) appear to be in

processing speed and aspects of sensory, verbal and working memory [2]. However, impairments are global and broad-based, also being reported across tests of attention, executive function, language, motor and spatial abilities, social cognition and general intelligence (i.e. around 1.0 pooled standard deviation) – indeed, on virtually *any* test of cognition administered. Such is the magnitude of these impairments, they equal or exceed those seen in composite cognition following moderate to severe traumatic brain injury [24], non-verbal memory following right-hemisphere stroke [25], executive impairments in attention-deficit hyperactivity disorder [26] and, perhaps most shockingly, memory impairments seen in preclinical and subsequent Alzheimer's disease [27]. On the basis of meta-analytic findings, 70-75 per cent of individuals with schizophrenia perform significantly below the general population across a wide range of cognitive tests [2]. In fact, Keefe *et al.* [28] have even reported that 98.1 per cent of individuals with a diagnosis of schizophrenia fail to reach their expected level of general cognitive ability (i.e. based on maternal education level). The cognitive deficits observed in schizophrenia seem to become apparent from around age 7 [29], with Gur *et al.* [30] reporting that, through the ages of 8 to 21 years, those who later go on to develop the disorder are between 6 to 18 months behind typically-developing children in terms of cognitive development. Consequently, Kahn and Keefe [1] have called for the re-conceptualisation of schizophrenia as *a primarily cognitive disorder*, with positive symptoms relegated to a secondary position.

Perhaps contrary to common perception, it seems that cognitive deficits cannot be attributed to the side-effects of anti-psychotic medication. In fact, longitudinal studies and meta-analyses suggest that anti-psychotics may have a beneficial, instead of detrimental, effect on cognition in general [31-34]. This notwithstanding, meta-analytic evidence *does* suggest that slow processing speed might be brought about, or at least augmented by, medication – possibly due to adverse effects on basal ganglia motor systems [35]. Impairments in spatial working memory have been reported in first-episode patients administered Risperidone [36], whilst different aspects of implicit memory have been found to be selectively impaired depending on whether first- or second-generation medications are administered [37]. Overall, though, it seems implausible that the range and magnitude of cognitive deficits in schizophrenia are a function of medication side-effects. Similarly, it seems unlikely that cognitive impairment results from the psycho-social stresses associated with a schizophrenia diagnosis. Firstly, moderate to severe impairments across most cognitive domains are

(already) present in first-episode patients [38, 39], as well as in an attenuated form in those identified as exhibiting prodromal symptoms [40]. Secondly, the deficit profile observed in such individuals is consistent with that seen in more chronic samples. Thirdly, the cognitive impairments observed in first-episode patients and prodromal individuals have been found to remain stable for up to 10 years [albeit with some deterioration in verbal memory; 41].

A growing literature indicates that cognitive impairments are an important predictor of functional outcome in schizophrenia [3]. Crucially, impairments in cognition appear to predict functional outcome to a greater degree than either positive or negative symptoms [42] – ‘functional outcome’ referring here not only to symptom-reduction and time spent in hospital, but also to factors such as degree of independence in community living, social relationships, occupational development and quality of life. Green [43] and Green *et al.* [3] reported medium-sized mean effect-sizes (i.e. $ES = 0.30$) for the relationship between individual cognitive abilities and functional outcome, whilst larger effect-sizes have been found between *composite* measures of cognition and functional outcome [44]. Specifically, Green *et al.* [3] reported that: (i) verbal fluency, executive functioning and secondary verbal memory (i.e. the ability to acquire and store verbal information from several minutes to much longer) reliably predicted community functioning; (ii) vigilance (i.e. sustained attention) and secondary verbal memory reliably predicted social problem-solving skills; and (iii) immediate and secondary verbal memory both reliably predicted psychosocial skill acquisition. A range of studies report robust associations between various cognitive domains and specific aspects of work functioning [e.g., 45, 46, 47]. Even when they have secured a job, those with the disorder often find it difficult to maintain employment, due to, for example, poor work performance [i.e. fewer than 15-20% of those with schizophrenia are employed; 48, 49, 50]. A meta-analysis by Fett *et al.* [51] reported that the amount of variance in functional outcome accounted for by cognitive measures ranged from 4 per cent, in terms of attention measures predicting social outcomes, to up to 23 per cent, for measures of social cognition predicting community functioning. Although this may seem modest, consider that stress electrocardiograms account for around 5 per cent of the variance in coronary artery disease and mammogram results account for around 9 per cent of cancer morbidity at one-year follow-up [52]. Due to the early onset of the disorder and its long-term impairing effects on the ability to look after oneself, complete activities of daily living and work, schizophrenia has been identified as one

of the ten most globally burdensome illnesses to individuals and societies throughout the world [53].

The above notwithstanding, it is possible to ameliorate cognitive impairments and their functional consequences. A substantial research (and financial) effort has examined the possible benefits of pharmacological interventions for cognitive impairments. Such interventions have included the administration of first- and second-generation anti-psychotics alone, as well as pharmacological augmentation – that is, multiple medications with different targets – thus, anti-psychotics for positive symptoms, anti-depressants for negative symptoms, as well as medications aimed specifically at the cognitive deficits. However, overall, medications have been shown to be of little benefit [54, 55]. Cognitive Remediation Therapy (CRT) – an umbrella term that refers to a range of different, though related, interventions, with the shared aim of improving impaired cognition – on the other hand, *has* been shown to be effective. In practice, CRT engages the participant in a wide variety of tasks, often delivered by a therapist, that are repeated over and over again until the specific ability in question has improved. In a randomised controlled trial (RCT) including 85 participants, Wykes *et al.* [56] reported that 40 weeks of CRT led to durable improvements in working memory ability which, in turn, led to improvements in social functioning. Furthermore, several meta-analyses have reported CRT to bring about medium-to-large effect-size improvements across various domains of cognitive functioning [6, 57, 58], with only a limited amount of cognitive remediation (e.g., 5-15 hours) being sufficient to bring about improvements. In combination with psychiatric rehabilitation, CRT can promote improvements in functioning [54, 57, 59]. Also, patients report CRT-based interventions to be engaging and enjoyable, and they are associated with increases in self-esteem [60]. It was arguably surprising, therefore, that the most recent NICE guidelines, published in 2014, did not recommend the provision of CRT to address cognitive deficits. NICE concluded that there is ‘no consistent evidence that cognitive remediation alone is effective in improving the critical outcomes’ (p. 249). However, NICE incorporated relapse rates, rehospitalisation and mental state as part of their ‘critical outcomes’. Even the most enthusiastic proponents of CRT may be cautious about its potential for having an impact upon such outcomes, the primary aim of CRT being to improve functioning in a specific cognitive domain(s), as well as improving aspects of broader (e.g., work) functioning negatively impacted by the deficit(s) in question.

I.III) THE PRESENT STUDY

Given the above, it is surely important that Psychosis services are not only aware of, but also dedicate an appropriate level of resource to, the understanding and management of cognitive impairments in patients. The present study therefore set out to explore this in an NHS CMHT for Psychosis in the UK.

II. METHODS

II.I) DESIGN

The design utilised a mixed-methods approach. At baseline, quantitative (via closed questions) and qualitative (via open-ended questions) data were derived from individual structured interviews with multi-disciplinary staff members in a Community Mental Health Team (CMHT) for psychosis, with the aim of exploring their knowledge and awareness of cognitive impairments in schizophrenia. At the end of each of these interviews, staff members were asked to: i) identify (the number of) patients on their caseload whom they believed to exhibit cognitive impairment/s, and; ii) rate their confidence (from 0-10, with '10' being 'the greatest confidence possible') in being able to notice cognitive impairments in patients with schizophrenia.

The interviews took place prior to participants attending a teaching session (i.e. the intervention) on cognitive impairments in schizophrenia. Following the teaching session intervention, staff members were asked to again: i) identify (the number of) patients on their caseload they believed to exhibit cognitive impairment/s, and; ii) rate their confidence in being able to notice cognitive impairments, from 0-10; these quantitative aspects of the design were thus repeated-measures in nature.

II.II) PARTICIPANTS

All staff members within a Multi-Disciplinary Community Mental Health Team (CMHT) for psychosis participated in the study. There were 15 participants, aged 34 to 65, of which 11 were female. There were no inclusion/exclusion criteria, other than being a staff member within the team.

The study was given ethical approval by the relevant body in the NHS Trust within which the study took place (not named here in order to preserve confidentiality).

Participants gave verbal consent after being provided with both a verbal and a written outline of the study during a Business Meeting within the CMHT. Participants were assured of confidentiality and were informed that they could end their participation at any stage and have their data removed.

II. III) MATERIALS AND PROCEDURE

II.III.I) BASELINE: STRUCTURED INTERVIEW SCHEDULE TO EXPLORE KNOWLEDGE AND AWARENESS OF COGNITIVE IMPAIRMENTS IN SCHIZOPHRENIA

A structured interview schedule, developed by the authors, was used to explore staff members' knowledge and awareness of cognitive impairments in schizophrenia. The interview schedule consisted of the 3 broad sections detailed below and, in general, tended to take around 30-35 minutes to administer. With the exception of one participant (who withheld consent), all interviews were recorded via Dictaphone and subsequently transcribed verbatim.

Both quantitative (closed questions) and qualitative (open questions) data were collected via the interviews. The type of data collected via each question is given in brackets below.

II.III.I.I) SECTION A: DEMOGRAPHIC INFORMATION (ALL DATA QUANTITATIVE)

Section A was used to collect the following information: i) age; ii) gender; iii) ethnicity; iv) position; v) the length of time (in years) employed in current position, and; vi) the length of time employed within the Trust.

II.III.I.II) SECTION B: KNOWLEDGE OF COGNITIVE IMPAIRMENTS IN SCHIZOPHRENIA AND RELATED QUESTIONS

In this section, the participant was asked the following questions:

1. Can you name and briefly describe for me any cognitive impairments that you are aware of in psychosis and schizophrenia? (Quantitative data; details of the method via which this question was quantified are given below.)
 - i. Do you know roughly what proportion of people with psychosis and schizophrenia are estimated to exhibit cognitive impairments? (Quantitative data.)

- ii. Do you know what sorts of things can be done to help manage these cognitive impairments? (Quantitative data; details of the method via which this question was quantified are given below.)
2. Have you had any teaching or workshops in cognitive impairments in psychosis and schizophrenia? ('Yes'/'No' – quantitative data.)
3. How much discussion would you say there is in your team around cognitive impairments in psychosis and schizophrenia? (Qualitative data.)
4. If and when you notice that a patient on your caseload seems to be exhibiting cognitive deficits, do you know what the options are, in terms of referral? (Quantitative data; details of the method via which this question was quantified are given below.)
 - i. Around how many patients would you say you have referred in the last year? (Quantitative data.)
5. Overall, how confident do you feel, from 0 to 10 (with '0' being 'not at all confident' and '10' being 'the greatest confidence possible') in terms of noticing/recognising cognitive impairments in patients? (Quantitative data.)

II.III.I.III) SECTION C: IDENTIFICATION OF COGNITIVE IMPAIRMENTS IN PATIENTS CURRENTLY ON THE PARTICIPANT'S CASELOAD (ALL DATA QUANTITATIVE)

As part of this section, the participant was presented with a copy of their current caseload and was asked to identify patients they believed to exhibit cognitive impairments. The following data were collected: i) the number of patients on the participant's caseload; ii) the number of patients identified by the participant as exhibiting/potentially exhibiting cognitive impairments, and; iii) for each patient identified by the participant, a brief description of the nature of the cognitive impairment/s the participant believed the patient to exhibit. Only those patients on participants' caseloads across both baseline and teaching session intervention were included as part of the data.

Where participants identified a patient as experiencing cognitive deficits, but then invalidated their response by going on to discuss difficulties which were clearly *not* cognitive in nature (specifically, difficulties related to positive or negative symptoms),

these patients were not included as part of the number of patients that participant had identified. In cases where patients were identified but their difficulties were not elaborated upon, participants were given the benefit of the doubt and they received a score for these participants. In cases in which participants discussed difficulties that were ambiguous, in terms of whether or not they referred specifically to cognitive impairments, participants were given the benefit of the doubt and scored positively. Thus, overall, participants were generally given the benefit of the doubt in cases of ambiguity.

Quantifying responses to Question 1 in Section B: Although this question was open-ended, participants' responses were quantified via systematic scoring criteria developed by the authors (See Appendix 1). Firstly, on the basis of meta-analytic literature reporting on the magnitude and range of forms of cognitive impairment in schizophrenia [cf. 18, 19-23], the following list of 9 broad cognitive domains, on which those with schizophrenia are shown to reliably perform worse than healthy controls, was drawn-up:

- i. Attention/concentration
- ii. Executive functions
- iii. General intelligence/IQ
- iv. Language abilities
- v. Memory
- vi. Motor skills
- vii. Processing speed
- viii. Social cognition
- ix. Visuospatial skills

Participants received a score of '1' for each form of cognitive impairment that they referred to (regardless of *how many times* they referred to this domain) *throughout the entirety* of the interview (i.e. not just in response to Section B, Question 1) – in order that they were given the best-possible chance to score. In response to Question 1, therefore, each participant received a score from 0 to 9. Participants' responses were scored independently by two researchers (MM and AC), with Kohen's kappa [61] used to examine inter-rater reliability. Kohen's kappa [61] can range between -1 to 1. Landis and Koch [62] characterise a kappa value of <0 as indicating no agreement, with values

between 0-0.20 indicating 'slight agreement', 0.21-0.40 as 'fair', 0.41-0.60 as 'moderate', 0.61-0.80 as 'substantial' and 0.81-1 as indicating 'almost perfect agreement'.

Quantifying responses to Questions 1, ii and 4 in Section B: These questions were open-ended but quantified categorically. Two researchers (MM and AC) independently produced initial codes to categorise participants' responses to each of these questions. The researchers then discussed their initial codes, before finalising the response categories.

II.IV) INTERVENTION: TEACHING SESSION ON COGNITIVE IMPAIRMENT/S IN SCHIZOPHRENIA

The teaching session intervention consisted of an approximately one-hour-long PowerPoint presentation to all MDT staff during a Business Meeting at the CMHT (see Appendix 2 for the PowerPoint slides). All staff members were provided with a copy of the slides in the form of paper hand-outs during the teaching session (and were subsequently sent the electronic version via email).

The presentation contained much of the information included in the Literature Review, above, and was divided into 3 sections. Broadly, the first, didactic section (slides 3-13) was concerned with:

- i. Defining the various domains of cognition known to be impaired in schizophrenia (slides 3-6)
- ii. A brief history of the concept of cognitive impairments in schizophrenia (slide 7)
- iii. Describing the magnitude and range of cognitive impairments in schizophrenia, as well as the proportion of those with schizophrenia estimated to exhibit cognitive impairments (slide 8)
- iv. Evidence that cognitive impairment/s in schizophrenia do not seem to be a function of either anti-psychotic medication or the stress associated with a diagnosis of schizophrenia (slides 9-10)
- v. The idea that, and mechanisms via which, cognitive impairment/s may affect functional outcome in schizophrenia (slides 11-12)
- vi. Evidence-based interventions to help manage cognitive impairments in schizophrenia, with a particular focus on Cognitive-Remediation Therapy (CRT) (slide 13)

In the second, more interactive section of the teaching session intervention, a series of slides presented some of the common ways in which certain aspects of cognitive impairment might manifest in everyday life (slides 14 to 20). After these slides had been presented, participants were each provided with a copy of their caseload and were given around five minutes to indicate (by marking against their names) any patients they believed to be exhibiting or possibly exhibiting cognitive impairment/s.

The third section of the teaching intervention (slides 21-26) consisted of a series of slides presenting simple, everyday strategies that could be used, by both patients (and their carers) and MDT members, in order to mitigate against cognitive impairments.

At the end of the session, all participants again rated, on a scale from 0-10 ('0' = 'not at all confident'; '10' = 'the greatest confidence possible'), their confidence in being able to recognise cognitive impairments in patients with schizophrenia. Participants were also asked to provide any feedback that they had following the presentation. Following the presentation, photocopies were taken of all participants' marked-on caseloads, with the originals being left with participants. This was done so that staff members: 1) could each see, quickly and easily, which of their patients may be exhibiting cognitive impairment/s, and; 2) could implement the mitigation strategies, discussed during the teaching session and printed on the hand-outs, corresponding to each patient's suspected cognitive impairment/s.

Due to other commitments, four staff members that had been interviewed at baseline were unable to attend the teaching intervention session. These people were therefore followed-up individually, presented with slides 14 to 20, provided with a copy of their caseload and taken through the same procedure as occurred during Section 2 of the teaching session – as well as being asked to re-rate their confidence and provide any feedback.

II.V) ANALYSES

II.V.I) QUANTITATIVE DATA

Simple descriptive statistics were used to explore the quantitative data generated via the baseline structured interviews.

Repeated-measures *t*-tests were used to explore whether: 1) participants' self-reported confidence in being able to notice cognitive impairments in their patients (rated 0-10) changed between baseline and post-intervention, and; 2) the numbers of patients identified by participants as exhibiting or possibly exhibiting cognitive impairments changed between baseline and post-intervention.

The number of participants that provided data for each of the quantitative analyses varied (e.g., due to some participants not having caseloads, participants not being contactable across both baseline and post-teaching time-points, etc.). The number of participants included as part of each of the quantitative analyses is stated in the description/outcome for each analysis presented in the Findings section.

II.V.II) QUALITATIVE DATA

Inductive thematic analyses, based on the 6-stage method described by Braun and Clarke [63], were used to analyse participants' responses to each of the two qualitative questions in the baseline interview. First, the data were transcribed (by MM). Following this, in order to allow familiarisation with the data and to facilitate initial analytic thoughts regarding meanings and patterns, the data were repeatedly read independently by two researchers (MM and AC). Second, the two researchers independently produced lists of initial codes manually from the raw data. The two researchers then compared and discussed their respective codes, before, thirdly, sorting them into a set of potential themes (with all the relevant coded extracts collated within the identified themes). Following this, themes were subjected to systematic initial refinement (e.g., both researchers discussed whether identified themes were indeed such, collapsing themes into each other, etc.) and further refinement, before being named. Finally, the results of the thematic analysis were written-up.

III. FINDINGS

III.1) DEMOGRAPHICS

Fifteen participants (i.e. all members of the CMHT) took part in the study. Participants' demographic characteristics are shown in Table 1. Unless otherwise stated, all 15 participants provided data in response to each question.

Table 1: Demographic characteristics of the complete sample of participants

Variable	N (Total = 15)	%
<i>Age (years)*</i>		
18-24	0	0.00
25-34	1	7.14
35-44	2	14.29
45-54	3	21.43
55-64	7	50.00
65+	1	7.14
<i>Gender</i>		
Female	11	73.30
Male	4	26.70
<i>Ethnicity</i>		
White: English / Welsh / Scottish / Northern Irish / British	8	53.33
White: Any other White background	1	6.67
Mixed / Multiple ethnic groups: White and Black Caribbean	1	6.67
Asian / Asian British: Any other Asian background	1	6.67
Black / African / Caribbean / Black British: African	3	20.00
Black / African / Caribbean / Black British: Caribbean	1	6.67

Table 1 continues over the page

Table 1 continued

Variable	N (Total = 15)	%
<i>Position</i>		
Support Worker	1	6.67
Social Worker	4	26.67
Community Psychiatric Nurse	6	40.00
Occupational Therapist	2	13.33
Specialist Registrar	1	6.67
Consultant Psychiatrist	1	6.67
<i>Length of time employed in current position</i>		
1-5 years	3	20.00
6-10 years	6	40.00
11-15 years	3	20.00
16-20 years	1	6.67
21-25 years	1	6.67
26+ years	1	6.67
<i>Length of time employed within Trust sites†</i>		
1-5 years	0	0.00
6-10 years	5	33.33
11-15 years	4	26.67
16-20 years	1	6.67
21-25 years	2	13.33
26+ years	3	20.00

* One participant declined to give their age and was therefore not included in the percentage calculations for age.

† The term 'Trust sites' is used here, rather than 'the Trust' as 4 participants were Social Workers and were therefore employed by the local authority, rather than by the NHS Trust.

The mean average age of participants was 52.50 ($SD = 8.50$) years. Participants had worked within the team for a mean average of 11.53 ($SD = 8.63$) years, and within Trust sites for 16.20 ($SD = 8.27$) years.

III.II) SECTION 1: DATA FROM PRE-INTERVENTION INTERVIEWS

The data presented and described in this section all relate to questions contained within Section B of the interview schedule.

QUESTION 1) 'CAN YOU NAME AND BRIEFLY DESCRIBE ANY COGNITIVE IMPAIRMENTS THAT YOU ARE AWARE OF IN PSYCHOSIS/SCHIZOPHRENIA?'

INTER-RATER RELIABILITY FOR SCORING CRITERIA

Table 2 presents the percentage agreement between the two raters and Kohen's kappa for each of the nine cognitive domains.

Table 2: Per cent agreement between the two raters and Kohen's kappa value for each cognitive domain, as well as overall mean

Cognitive domain	Agreement (%)	Kohen's kappa
<i>Attention/concentration</i>	92.9	0.86
<i>Executive functions</i>	92.9	0.76
<i>General intelligence</i>	78.6	0.51
<i>Language</i>	71.4	0.44
<i>Memory</i>	92.9	0.76
<i>Motor skills</i>	92.9	0*
<i>Processing-speed</i>	92.9	0.76
<i>Social cognition</i>	85.7	0.44
<i>Visuospatial skills</i>	100	1
<i>Overall mean</i>	88.91	0.69†

* Kohen's kappa could not be computed due to rater 1 not scoring any participant positively for this domain, meaning that this variable was a constant.

† Value calculated without Kohen's kappa for motor skills.

As can be seen in the Table, percentage agreements were all relatively high. The majority of kappa values (5/8) fell into either the 'substantial' or 'almost perfect agreement' ranges, with the remaining three falling into the 'moderate' range [62]. The mean kappa value fell into the 'substantial agreement' range. Good levels of inter-rate reliability were therefore achieved for the scoring criteria.

NUMBER OF COGNITIVE DOMAINS IDENTIFIED BY PARTICIPANTS

Given the high levels of inter-rater reliability achieved, the two raters' scores were mean-averaged. The mean numbers of participants that identified the various cognitive domains often impaired in schizophrenia, as scored by the two raters, are presented in Table 3.

Table 3: Mean numbers and percentages of participants that identified the various cognitive domains impaired in schizophrenia ($N = 15$)

Cognitive domain	Number of participants that identified domain	Percentage of participants that identified domain
<i>Attention/concentration</i>	7.5	50.00
<i>Executive functions</i>	12.5	83.33
<i>General intelligence</i>	4.5	30.00
<i>Language</i>	7	46.67
<i>Memory</i>	12.5	83.33
<i>Motor skills</i>	0.5	3.33
<i>Processing speed</i>	2.5	16.67
<i>Social cognition</i>	2	13.33
<i>Visuospatial skills</i>	1.5	10.00

The mean number of domains identified by *each individual participant* was 3.36 ($SD = 1.20$), out of a possible 9. The table shows that memory and executive functions were clearly the domains identified most often by participants. Indeed, these domains were identified around twice as often as the next-most-identified domains of attention/concentration and language. Notably, very few participants identified impairments in motor or visuospatial skills.

Almost half the participants (7) conflated the positive symptoms of schizophrenia with cognitive impairments at some point in their naming/describing the various cognitive domains, as illustrated by the following:

They [patients] cannot understand... something, something real, whether they, maybe they're hearing voices or they're seeing things that are not there, and they can't put it together to say, 'Is this real or not real?' So I would say that they haven't got the knowledge of what's

going-on around them – so that’s my understanding of cognitive impairment in schizophrenia (Participant 13).

One participant equated the negative symptoms with cognitive impairments.

QUESTION 1. I) ‘DO YOU KNOW ROUGHLY WHAT PROPORTION OF PEOPLE WITH PSYCHOSIS/SCHIZOPHRENIA ARE ESTIMATED TO EXHIBIT COGNITIVE IMPAIRMENTS?’

Where participants gave an upper and a lower estimate in response (e.g., ‘30-40 per cent’), their response was scored as the mid-point between these two values (e.g., ‘35%’). The mean percentage of patients with schizophrenia estimated by participants to show cognitive impairment/s was 53.45 ($SD = 25.47$); the range of estimates here was substantial, however, from 10 to 100 per cent. Nevertheless, it is true that a number of participants’ estimates clustered around the 50 per cent mark, with 7 participants estimating within or below 15 per cent of the 50 per cent mark. This mean estimate is substantially lower than the approximately 70-75 per cent of patients thought to demonstrate cognitive impairment [2].

QUESTION 1. II): ‘DO YOU KNOW WHAT SORTS OF THINGS CAN BE DONE TO HELP MANAGE COGNITIVE IMPAIRMENTS IN PSYCHOSIS/SCHIZOPHRENIA?’

Table A1 in Appendix 3 presents the forms of intervention strategy for cognitive impairments in schizophrenia mentioned by participants, together with illustrative quotations and the numbers of participants that mentioned each strategy. Participants were free to mention any number of strategies. Thus, participants mentioned a number of possible interventions, although only two described Cognitive Remediation Therapy (CRT) – the approach with the best evidence-base [6, 56-59]. Each participant mentioned a mean of 2.73 ($SD = 1.62$; $range = 0-6$) possible interventions.

QUESTION 2: 'HAVE YOU HAD ANY TEACHING OR WORKSHOPS IN COGNITIVE IMPAIRMENTS IN PSYCHOSIS/SCHIZOPHRENIA?'

Perhaps surprisingly, not a single participant had had any specific teaching/training concerning cognitive impairments in schizophrenia.

As a follow-up to the question of whether participants had had any specific training on cognitive impairments in schizophrenia, 11 participants were asked whether they would like to learn more about this area; four participants were not asked this question (due, for example, to their having spontaneously stated their lack of teaching/training at a point in the interview before they were due to be asked by the researcher, and the researcher's subsequently failing to return to this issue at what would have been the appropriate part of the interview). Of the eleven participants asked, 100 per cent said that they would like to learn more about cognitive impairments in schizophrenia. Indeed, the enthusiasm for teaching/training in this area shown by participants is illustrated by Participant 1, who, in response to being asked whether they would like to receive such training, said: 'Yes! Definitely, definitely... I think, definitely, we need more training around that.'

QUESTION 3: 'HOW MUCH DISCUSSION WOULD YOU SAY THERE IS IN YOUR TEAM AROUND COGNITIVE IMPAIRMENTS (IN PSYCHOSIS/SCHIZOPHRENIA)?'

As part of their responses to this question, 11/15 (73%) participants spontaneously stated, in various ways (e.g., 'I'm not aware that there's that much' [Participant 4], 'I think we touch on it... It's not really something that we talk about' [Participant 11]), that discussion of cognitive impairments in schizophrenia in the team was limited.

Thematic analysis of participants' responses revealed 2 key themes: i) Attenuated awareness and discussion due to resources and ii) Discussion occurs as and when a serious/obvious impairment presents itself.

Attenuated awareness and discussion due to lack of resources

Around half of participants (7/15) expressed the view that awareness and discussion of cognitive impairments were attenuated due to various resources being limited or unavailable within the service: 'I think a lot is dep-, er, depending on the resources you have, as well, to address [this thing]' (Participant 2). Participants talked about the day-to-day demands of their job making it difficult to find the time to first consider

cognitive impairments: 'We need to have more time to reflect about some of the challenges, rather than trying to respond all the time...' (Participant 1). Related to this, there was also a feeling that the often-stressful nature of the job – specifically, and ironically, interactions with patients possibly affected by cognitive impairments – can in itself reduce the chances that clinicians may recognise these possible cognitive impairments in patients: 'You know, often we can be frustrated ourselves, as clinicians, you know, "Why can't people see the logic in this matter?", when maybe we should be thinking, "Well, what is [sic] the barriers for people being able to do [that]?" You know, "Is it their choice that they're not able to do [that]...?"' (Participant 1). One participant vividly described how the assessment tools utilised by the service (and, of course, by services in general) do not allow for the consideration of cognitive impairments in schizophrenia: 'Where we might get new referrals, in fact, I'm, there's probably nothing in the integrated assessments around [that]' (Participant 12). Interestingly, and owing to participants' perceived lack of resources in their service to address cognitive impairments, there was an indication that the lack of consideration and discussion may be wilful: 'But I don't think we, well, I, I... maybe that we don't have enough skills or materials to help [them] overcome [that], so therefore, we, we don't pay enough attention to [it] – because if we can't repair something, what's the use of, erm, peering over it for so long?' (Participant 8). It also seemed that some participants believed that the consideration of cognitive deficits fell under the purview of specific roles within the team, and was therefore not a general concern: 'I don't know, maybe, maybe we expect doctors to, to be assessing [that]...' (Participant 12). Relatedly, a lack of knowledge/expertise in being able to recognise cognitive impairments was also highlighted: 'I wouldn't, I don't really feel very confident, I just, sort of, might make an observation, erm, you know, having worked with somebody, but yeah, I wouldn't, I wouldn't feel that comfortable maybe pinning it down in an assessment' (Participant 12).

Discussion occurs as and when a serious/obvious impairment presents itself

Seven participants said that, though they are not a part of routine discussion, cognitive impairments are talked about when patients' difficulties are clearly noticeable: 'I'm not sure we specify that there is cognitive impairment... unless the person is presenting with very severe deficits' (Participant 3). Some participants elaborated further, suggesting that such discussions tend to be restricted to non-schizophrenia-related

cognitive issues. For example, Participant 15 said: ‘... A few team meetings with, erm, about certain concerns about certain clients, but again, that was more around, “Are we concerned that someone’s gaining / has developed a dementia? And what [sic] services?”, rather than ... related to psychosis’. Similarly, Participant 12 said the following: ‘I think we talk, we think about it sometimes, when we, you know, there’s substance misuse and things.’ However, although discussion may indeed occur, participants indicated that such talk tends to occur in a rather informal, implicit manner, as opposed to an explicit and distinct aspect of team discussions:

Most of the discussion, I guess, I would hear around cognitive impairments would just be people commenting, perhaps, o-, on, on, or maybe giving a scenario of something they’ve experienced, one of their own clients and, and just having a conversation about, say, how maybe difficult a particular encounter is, or whatever, so, erm, so it-, it-, that’s kind of, making-up a part of a conversation, rather than any kind of *formal* discussion, yeah, if you see what I mean, yeah (Participant 4).

QUESTION 4) ‘IF AND WHEN YOU NOTICE THAT A PATIENT ON YOUR CASELOAD SEEMS TO BE EXHIBITING COGNITIVE DEFICITS, DO YOU KNOW WHAT THE OPTIONS ARE, IN TERMS OF REFERRAL?’

Table 6 presents the categories into which responses fell, together with the numbers of participants whose responses fell into each category. Participants were free to mention any number of referral options.

Table 6: Participants' referral routes / discussion fora for patients with cognitive impairment and the numbers of participants whose responses fell into each category
($N = 15$)

Response category	Number of participants that gave response
<i>Internal:</i>	
<i>Discuss with Care Coordinator</i>	2
<i>Discuss with other MDT members</i>	2
<i>Discuss with Occupational Therapist (OT) / Refer for OT assessment</i>	2
<i>Discussion with / referral to Psychiatrist</i>	3
<i>Discussion with / referral to Psychology (unspecified)</i>	3
<i>Psychometric assessment</i>	3
<i>Mindfulness Group</i>	2
<i>Discussion with Supervisor</i>	3
<i>Unspecified within-team referral</i>	3
<i>External:</i>	
<i>Autism Spectrum Disorders (ASD) Service</i>	1
<i>Signpost to General Practitioner (GP)</i>	1
<i>Memory service (for dementia assessment)</i>	1
<i>Neurology (if learning disability; LD)</i>	1
<i>Neuropsychiatry</i>	1
<i>Neuropsychology</i>	1
<i>Support agencies (unspecified)</i>	1
<i>Don't know</i>	5

The mean number of referral routes mentioned by each participant was 1.73 ($SD = 1.22$; range = 0-4). As part of their responses to the question, most participants (12/15) mentioned intra-team referrals / discussions. Six participants mentioned external referrals, with 3 routes being related to cognitive impairments in the context of other disorders (ASD, dementias and LD), with signposting to GP and referral to unspecified support agencies also being mentioned. External referrals to either Neuropsychiatry or

Neuropsychology were each mentioned by one participant. As part of their responses to this question, 5 participants said that they ‘don’t know’.

QUESTION 4. I): ‘AROUND HOW MANY PATIENTS WOULD YOU SAY YOU HAVE REFERRED, ON THE BASIS OF COGNITIVE IMPAIRMENTS, IN THE LAST YEAR?’

Thirteen participants contributed data here (i.e. all but two who did not have a caseload). The mean percentage of patients that each participant reported (as a proportion of their caseload) having referred in the last year, on the basis of their presenting with cognitive impairments, was 14.15 ($SD = 15.31$). The range of scores was considerable, with 4 people reporting not having referred any of the patients on their caseload within the last year, whilst the highest self-report was 50 percent. Relatedly, it appeared that the relatively small mean quoted above was nevertheless inflated by four participants self-reporting percentages of 27 and above; the remaining 9 participants reported percentages of 4 or lower.

Notably, one participant, who reported having referred 27 percent of their caseload, spontaneously volunteered that all of these referrals had been to the Mindfulness Group. Additionally, after having given their response to this question, Participant 14 (who had reported referring 30% of their patients) spontaneously said: ‘... On reflection-, I think that, erm, I mean there is a-, what is cognition? What do we mean by it? I don’t really know what we mean by it, so that’s why it’s difficult to actually answer the questions.’

III.III) SECTION 2: COMPARING DATA ACROSS PRE-INTERVENTION INTERVIEWS AND POST-TEACHING INTERVENTION

CONFIDENCE IN NOTICING/RECOGNISING COGNITIVE IMPAIRMENT/S IN SCHIZOPHRENIA IN PATIENTS PRE- AND POST-INTERVENTION

Participants’ mean self-report ratings for their confidence in being able to notice/recognise cognitive impairment/s in Schizophrenia, at both pre- and post-intervention, are given in Table 7. There were 14 participants with complete data here (one participant could not be contacted for their rating post-intervention).

Table 7: Participants' ($N = 14$) mean (SD) self-reported confidence in noticing/recognising cognitive impairment/s in schizophrenia at pre-intervention interview and post-intervention

Mean self-reported confidence in noticing/recognising cognitive impairment/s at pre-intervention interview*	Mean self-reported confidence in noticing/recognising cognitive impairment/s post-intervention*
5.96 (2.06)	7.18 (1.49)

* Participants were asked to rate their confidence on a scale from 0 to 10, anchored by the statements 'Not at all confident' (0) and 'The greatest possible confidence' (10).

A repeated-measures t -test found that the greater confidence in noticing cognitive impairments in schizophrenia reported by participants at post-intervention was statistically significant [$t(13) = -2.49$; $p = 0.03$, 2-tailed].

IDENTIFICATION OF PATIENTS ON PARTICIPANTS' CASELOADS PRE- AND POST-INTERVENTION

The mean percentages of patients that each participant identified on their caseload as demonstrating cognitive impairment/s during the pre-intervention interviews and post-intervention are presented in Table 8. Percentages are given here, rather than the absolute numbers of patients identified by participants, due to each participant having a different number of patients on their caseload. Four participants did not provide complete data at both pre-intervention interview and post-intervention: two participants did not have caseloads and another 2 participants provided data at pre-intervention interview but not at post-intervention. Thus, 11 participants had complete data for these analyses.

Three participants described problems unrelated to cognitive impairment (e.g., positive symptoms, negative symptoms, other difficulties, etc.) when identifying certain patients on their caseloads; these patients were therefore not included in the data.

Table 8: Mean percentages (*SD; range*) of patients that each participant identified on their caseload as showing cognitive impairment/s at pre- and post-intervention ($N = 11$)

Mean percentage of patients identified at pre-intervention interview	Mean percentage of patients identified at post-intervention
33.24 (19.84; 0-70.59)	80.64 (22.49; 27.27-100.00)

A repeated-measures *t*-test found that the greater percentage of patients identified by participants at post-intervention was highly statistically significant [$t(10) = -9.04$; $p < 0.01$, 2-tailed].

IV. DISCUSSION/IMPLICATIONS OF THE STUDY

Overall, the results from the present study are consistent in indicating that, within the CMHT, staff members do not generally actively look-out for cognitive impairments in schizophrenia, nor are they systematically addressed. The main findings were that: i) participants identified a mean number of 3, from a possible list of 9, cognitive impairments often exhibited by those with schizophrenia; ii) on average, participants (under)estimated that around 53 per cent of individuals with schizophrenia exhibit some form of cognitive impairment; iii) only 2/15 participants were aware of Cognitive Remediation Therapy (CRT) as an empirically-supported intervention for cognitive impairment; iv) not a single participant had received any specific teaching/training concerning cognitive impairments in schizophrenia; v) 73 per cent of participants indicated that there was an inadequate amount of discussion of cognitive impairments in schizophrenia, with lack of resources highlighted as a key factor, and; vi) within the previous year, participants had referred only around 14 per cent of the patients on their caseloads, on the basis of their exhibiting cognitive impairments. Additionally, participants rated their baseline confidence in being able to notice/recognise cognitive impairments in patients at a mean of 6 (out of 10), which (statistically-significantly) increased to 7 (out of 10) post-teaching-session. Finally, at baseline interview, participants identified an average of 33 per cent of the patients on their caseloads as exhibiting cognitive impairments, which subsequently (statistically-significantly) increased to 81 per cent of patients post-intervention.

IV.1) LIMITED KNOWLEDGE AND AWARENESS OF COGNITIVE IMPAIRMENT IN SCHIZOPHRENIA

That participants described a mean average of only a third of the cognitive impairments often observed in schizophrenia suggests that, in daily practice, MDT staff members are not familiar with most of the impairments presumably being displayed by patients. Furthermore, at some point during their interviews, almost half of participants conflated the positive symptoms of schizophrenia with cognitive impairments. This demonstrates a lack of understanding regarding the nature of cognitive impairments – specifically, their status as an independent category of difficulty exhibited in schizophrenia, fundamentally separate from hallucinations and delusions. The two domains of cognitive impairment most mentioned (by some distance) by participants were memory problems and executive functioning difficulties. Although these are important cognitive difficulties commonly observed in the disorder, the impairment with the largest effect-size [on the basis of meta-analytic studies; 2] – (slow) processing speed – was referred to by just 3 (of 15) staff members. Similarly, impairments in social cognition – for which large effect-sizes have also been reported [23] – were also mentioned by only 3 staff members.

A potential limitation of the study was that the baseline interviews were generally ‘squeezed in’ as and when members of staff had ‘gaps’ in their diary, between sessions with patients. It could be argued that, under these circumstances, participants were not able to focus and bring to mind and articulate the extent of their knowledge around cognitive impairments, and that the present results are therefore an under-representation of their knowledge in this area. In response to this, it is argued that, in the context of a busy CMHT, staff members’ time is a precious commodity and it is thus incredibly difficult to conceive of an alternative means via which these data could have been collected. In fact, that the present study managed to recruit all members of the MDT, giving them all the opportunity to demonstrate their awareness of cognitive impairments, despite these substantial time-pressures, was a significant strength. Also, and as stated in the Methods section, participants’ referencing of each of the listed domains of cognitive impairment was scored across the *whole interview*, not just in response to the first question (which asked explicitly about this). This, together with the fact that, generally, baseline interviews lasted around 30-35 minutes, meant that participants were given time to gather themselves, ease into the interview and

gradually call to mind their knowledge of cognitive impairments in schizophrenia, giving them the best chance of demonstrating their knowledge as was feasible. Furthermore, and as can be seen in Appendix 1, the scoring system was relatively liberal, such that participants scored positively for a domain if they gave, 'any clear reference to, or unambiguous examples of, difficulties in [that area]', rather than having to explicitly name the domain to which they were referring. Finally, the total number of potential domains to which participants could refer was reduced as far as possible, such that, for example, short-term and long-term forms of memory were incorporated within a single domain. Overall, therefore, a substantial effort was made to 'tap into' and represent the 'true' extent of participants' knowledge.

During the baseline interviews, participants estimated that 53 per cent of those with schizophrenia exhibit cognitive impairments. This is substantially lower than the 70/75-98 per cent of patients thought to demonstrate cognitive impairment [2, 28] and thus constitutes an underappreciation of the extent to which cognitive impairments affect those with schizophrenia. Similarly, participants identified an average of just 33 per cent of the patients on their caseloads as exhibiting cognitive impairments – some way short of the 70-75 per cent of individuals with schizophrenia estimated, on the basis of meta-analytic findings, to show such difficulties [18-23]. However, a limitation of the present study was that it was not feasible to determine the 'true' number of patients showing cognitive impairments on the caseload of the CMHT (by, for example, assessing patients). Then again, the fact that such a figure would have had to have been generated by the present researchers as part of the study, rather than being accessed via records held as a routine part of the service, might in itself be noteworthy. Regardless, this means that it is not possible to conclude that any patients with cognitive impairments had been 'missed' by participants at the baseline interviews. This notwithstanding, post-teaching-intervention, participants identified a significantly greater 81 per cent of their patients as exhibiting cognitive impairments – the proximity of this figure to estimates derived from meta-analytic studies being notable. Accordingly, participants also rated their self-confidence in being able to detect/recognise cognitive impairments as being significantly higher following the intervention.

Taken together, the above suggests that staff members in the current MDT are to a large extent unaware of the forms and extent of cognitive impairments in

schizophrenia. The extent to which these findings are representative of other MDTs in the country is unclear, and so similar studies in other teams would be useful. Nevertheless, given the potentially widespread impact that such problems can have on an individual's functioning [3, 43-44], a lack of awareness of this aspect of patients' presentations could lead to difficulties. Suppose, for example, that slow processing speed meant that, in a meeting with a clinician, it takes a patient a significantly longer amount of time, firstly, to interpret and understand that which their clinician says to them/asks them, and secondly, to formulate an appropriate verbal response. This would tend to have the consequence that, overall, the patient might not end-up saying very much in the session(s). If the clinician is unaware of this, then it might be reasonable for them to conclude – albeit incorrectly – that the patient did not wish to answer the question, was not listening to what they had said or had nothing to say on that particular subject. The clinician might abandon the topic and move-on to something else, when, in reality, the patient had simply not had enough time to articulate their response. It is important to state that a given score indicating impairment in a given cognitive construct, in a neuropsychological assessment, does not purely and simply translate to a given list of observable manifestations related to that impairment. Nevertheless, if clinicians are unaware that such difficulties are often present in schizophrenia, clinicians will understandably tend not to adapt their practice with patients on the basis of such impairments (e.g., allowing the patient more time to formulate responses to questions/statements). This would be expected not only to impact on the relative 'success' of clinicians' sessions with patients, but also more broadly: if the underlying cognitive impairments are not themselves recognised and addressed, this may compromise the extent to which patients are able to pursue and maintain recovery goals [3, 43-44].

Given that cognitive impairments are considered fundamental in Schizophrenia [1], it is perhaps surprising that not a single participant had had any specific teaching/training concerning these issues. This almost certainly explains participants' relative lack of awareness of cognitive impairments in patients: people cannot be blamed for being broadly unaware of issues in which they have had no formal training and to which the wider system directs such little attention.

IV.II) LIMITED DISCUSSION OF AND REFERRAL FOR COGNITIVE IMPAIRMENTS IN SCHIZOPHRENIA

Eleven out of fifteen percent of participants felt that discussion of cognitive impairments in the team was limited, which they related to a lack of resources – namely, a lack of knowledge/awareness of what to look-out for, combined with the ongoing demands (and stress) of staff members' jobs, a lack of time and an absence of materials to screen for and address potential cognitive impairments. Participants described discussing cognitive impairments as and when they were so severe as to be a salient aspect of a patient's presentation – and often in a non-schizophrenia context, such as in cases of a suspected dementia or when related to substance abuse. Participants said that, when these discussions do occur, they tend to be 'informal' and 'by-the-by' in nature, rather than being formally and systematically explored and problem-solved by the team. Overall, participants' comments suggest an endemic lack of awareness and consideration of cognitive impairments within Psychosis Services.

When asked about possible interventions for cognitive impairments, a wide range of potential approaches were described by participants, although many of these would not traditionally be expected to directly address any underlying deficits (e.g., attending social groups, encouraging independence, engagement in meaningful activity, improving sleep hygiene, mindfulness, having a Support Worker, increasing insight around positive symptoms), thereby suggesting a lack of understanding concerning the nature of cognitive impairments in schizophrenia. Four participants mentioned possible interventions that would appear reasonable from a lay perspective but which are questionable according to the available evidence – such as, firstly, medication to act directly on impairments and, secondly, managing the side-effects of anti-psychotic medication. Medications to directly act upon cognitive impairments appear ineffective [54, 55] and anti-psychotic medication has a mildly beneficial, rather than harmful, effect on cognition [31-34]. This notwithstanding, staff members' responses suggested considerable effort in trying to address/ameliorate (the impact of) cognitive impairments using the limited means at their disposal. Specifically, two-thirds of participants (and over 20% of total responses) mentioned practical, Occupational Therapy-type adaptations (e.g., phone alarms, diaries) as a way of managing cognitive impairments. Similarly, 27% of participants also spoke about the need to be person-centred, looking at the (cognitive) strengths and weaknesses of any given patient. Only

two participants mentioned Cognitive Remediation Therapy (CRT) – the approach with the strongest evidence-base [6, 56-58]. This appears understandable, given that the only referral route for cognitive impairments in schizophrenia in the CMHT was to the Clinical Psychologist for a cognitive assessment – with no further routes onward for CRT if/when cognitive impairments are detected. Then again, the latest NICE guidelines for Psychosis (2014) do not recommend the provision of CRT, meaning that the CMHT was merely adhering to national guidelines.

Given the lack of an external referral route when cognitive impairments are suspected and/or detected, it is unsurprising that two-thirds of responses to the question of referral routes used by MDT members described within-team referrals / discussions with other team members. Only 3/15 participants said that they would refer to the Clinical Psychologist for a cognitive assessment (in accordance with the designated referral route for cognitive impairments in the service). Overall, given participants' comments on the lack of resource to address cognitive impairments in the service, the potential effectiveness of these inter-team referrals and their associated interventions is surely questionable. It is also notable that 4/15 participants (and around 11% of total responses) mentioned external referral pathways that had no clear relevance to cognitive impairments in schizophrenia; that is, referrals to: external Autism Spectrum Disorders services, memory services (for dementia assessment) and neurology services (if presence of a learning disability); signposting to GP, and; referral to external 'support agencies'. Five (out of fifteen) participants (around 15% of total responses) said that they did not know where they could refer. It makes sense, given all of the above, that, within the year prior to baseline interview, participants had only referred 14% of their patients, on the basis of cognitive impairments: if staff members do not recognise cognitive impairments, and indeed underestimate their prevalence, as well as not having a clear referral route to adequately address such problems, this clearly reduces the chances of their making referrals. Regardless of the underlying reasons, that only 14% of patients on the caseload of the CMHT were referred strongly suggests that the cognitive impairment that would be expected to be exhibited by a sizeable number of patients is not being adequately and systematically addressed, with potential implications for patients' functioning and wellbeing.

Overall, the data appear to suggest a team trying to identify and manage cognitive impairments in their patients as best they can under (highly) challenging

circumstances – that is, given their lack of teaching/training, the lack of specialist services to which patients can be referred for assessment/intervention and the lack of resource in Psychosis Services in general to meet the need for cognitive impairments in schizophrenia to be properly addressed.

An immediate and obvious argument against the training of MDT staff and provision of CRT would be the current economic climate. Specifically, according to recent estimates, the UK NHS is around £2.5 billion in debt (the Health Foundation, 2016), with real-term reductions in spending amounting to 1% for working-age adults and 3.1% for older adults (NHS Confederation, 2016) – with the funding shortfall projected to increase to at least £16 billion by 2030/2031 (the Health Foundation, 2016). Seen against this backdrop, the current lack of resource dedicated to cognitive impairments in schizophrenia might appear justified. Also, in fairness to those responsible for commissioning and mandating such training and services, the research evidence around cognitive impairments in schizophrenia, and the beneficial effects of CRT, has only relatively recently become established. Also, CRT is a relatively time- and resource-intensive process and may require dedicated staff working specifically on remediation. This notwithstanding, it is now clear that cognitive impairments exert a significant and substantial influence over patients' work, community functioning and broader recovery [3, 43-47, 51] – indeed, a significantly greater influence than either the positive or negative symptoms [42]. Schizophrenia is one of the ten most globally burdensome disorders in the world, for both individuals and societies [53] and it has been estimated that schizophrenia costs the UK economy £11.8 billion a year [64]. With only around 15-20% of those with a diagnosis employed [48], and measures of cognitive functioning having been shown to account for up to 23% of individuals' functional outcome [51], it can be reasonably assumed that a substantial proportion of the financial costs associated with schizophrenia are related to impairments in cognition. Participants in the present study identified a seemingly more valid proportion of the patients on their caseloads as exhibiting cognitive impairments, as well as self-reporting a greater level of confidence in being able to detect cognitive impairments, after just one teaching session. This suggests that the financial and time costs of providing training to identify these difficulties may not be particularly substantial – yet could lead to a significantly heightened awareness of cognitive impairments in schizophrenia within MDTs. Similarly, it is surely worth considering the potential financial and wider social benefits of setting-up and running specialised services for

delivering CRT (together with corresponding NICE guidelines), in order to address the many and varied financial and social costs associated with cognitive impairment in schizophrenia. Indeed, a continued lack of resource to address this huge issue appears difficult to justify.

IV.III) CONCLUSION

Overall, the present study found that, in a CMHT for psychosis, staff members had limited knowledge and awareness of cognitive impairments in schizophrenia. These findings are hardly surprising, given the current lack of resource (e.g., training, specialised services, etc.) allocated to cognitive impairments in schizophrenia. This should surely be re-evaluated in the light of growing evidence of the breadth and magnitude of cognitive impairments in schizophrenia, together with their important implications for functioning and general wellbeing.

V. DISSEMINATION

The findings of the project are to be presented to the team in the form of a PowerPoint presentation at one of their monthly Business Meetings. I also hope that it can be prepared, in whole or in part, as part of an application to commissioners for resource to address cognitive impairments in schizophrenia in Psychosis Services. This report will also be submitted as an article to peer-review journals, as well as being submitted for (poster or oral) presentation at conferences.

VI. LEADERSHIP

This project was carried-out at my first placement. I demonstrated leadership in the first instance by developing the rationale and protocol for the project and taking this to my placement supervisors, who agreed that it was worthwhile and that they would supervise it. As stated above, all staff members participated in the project, which involved their agreeing to a 30-35-minute structured interview at baseline, followed by their completing the post-intervention identification of patients with possible cognitive impairments on each of their caseloads. Clearly, this was relatively 'involved' for staff members in a busy CMHT and I believe that the good working relationships that I had developed and maintained with them whilst on placement helped facilitate their engagement in the project. In addition, staff members had often commented that they wanted to learn (more) about cognitive impairments in schizophrenia; that I was

offering a teaching session on this area probably also aided their participation. Staff members were often busy and had to reschedule interviews at the last moment (e.g., due to patient emergencies); I believe that my skills in being gently but firmly persistent in following-up staff members through such occasions was an important factor in my nevertheless managing to collect data from all of them. I led on all stages of the data-analysis and report write-up. I strongly believe and hope that the findings from this report can be used to bring about the provision of greater resource-allocation for cognitive impairments in schizophrenia.

REFERENCES

1. Kahn, R.S. and R.S. Keefe, *Schizophrenia is a cognitive illness: time for a change in focus*. JAMA Psychiatry, 2013. **70**(10): p. 1107-112.
2. Heinrichs, R.W., et al., *Cognition as a central illness feature in schizophrenia*, in *Cognitive Impairment in Schizophrenia: Characteristics, Assessment and Treatment*, P.D. Harvey, Editor. 2013, Cambridge University Press: The Edinburgh Building, Cambridge.
3. Green, M.F., et al., *Neurocognitive deficits and functional outcome in schizophrenia: are we measuring the "right stuff"?* Schizophr Bull, 2000. **26**(1): p. 119-36.
4. Association, A.P., *Diagnostic and Statistical Manual of Mental Disorders: DSM-5 (5th Ed.)*. 2013, Washington, D.C.: American Psychiatric Association.
5. Organization, W.H., *The ICD-10 classification of mental and behavioural disorders: clinical descriptions and diagnostic guidelines*. 1992: Geneva: World Health Organization.
6. Wykes, T., et al., *A meta-analysis of cognitive remediation for schizophrenia: methodology and effect sizes*. American Journal of Psychiatry, 2011. **168**(5): p. 472-485.
7. Excellence, N.I.f.H.a.C., *Psychosis and schizophrenia: treatment and management. (Clinical guideline 178)*. 2014.
8. Royall, D.R., et al., *Executive impairment among the functionally dependent: comparisons between schizophrenic and elderly subjects*. American Journal of Psychiatry, 1993. **150**: p. 1813-1813.

9. Pinkham, A.E., *Social cognition and its relationship to neurocognition*, in *Cognitive Impairment in Schizophrenia: Characteristics, Assessment and Treatment*, P.D. Harvey, Editor. 2013, Cambridge University Press: Cambridge.
10. Kraepelin, E., *Psychiatrie: ein lehrbuch für studierende und artze. (5th edition.)*. 1896, Leipzig: Barth.
11. Kraepelin, E., *Dementia praecox and paraphrenia* (R. M. Barclay, Transcriber). 1919, Edinburgh: Livingstone.
12. Bleuler, E., *Lehrbuch der Psychiatrie*. 1943, Berlin: Springer.
13. Bleuler, E., *Dementia praecox, or the group of schizophrenias. (J. Zinkin, Transcriber)*. 1950, New York, NY: International Universities Press. (Original work published 1911.).
14. Randolph, C., T.E. Goldberg, and D.R. Weinberger, *The Neuropsychology of Schizophrenia.*, in *Clinical Neuropsychology. (3rd Ed.)*, K.M. Heilman and E. Valenstein, Editors. 1993, Oxford University Press: New York, NY.
15. Wechsler, D., *WAIS-IV: Wechsler adult intelligence scale*. 2008: Pearson San Antonio, TX.
16. Shakow, D., *Segmental set*. Arch Gen Psychiatry, 1962. **6**: p. 1-17.
17. Shakow, D., *Psychological deficit in schizophrenia*. Behavioural Science, 1963. **8**: p. 275-305.
18. Heinrichs, R.W. and K.K. Zakzanis, *Neurocognitive deficit in schizophrenia: a quantitative review of the evidence*. Neuropsychology, 1998. **12**(3): p. 426-45.
19. Aleman, A., et al., *Memory impairment in schizophrenia: a meta-analysis*. Am J Psychiatry, 1999. **156**(9): p. 1358-66.
20. Johnson-Selfridge, M. and C. Zalewski, *Moderator variables of executive functioning in schizophrenia: meta-analytic findings*. Schizophr Bull, 2001. **27**(2): p. 305-16.
21. Forbes, N.F., et al., *Working memory in schizophrenia: a meta-analysis*. Psychol Med, 2009. **39**(6): p. 889-905.
22. Mesholam-Gately, R.I., et al., *Neurocognition in first-episode schizophrenia: a meta-analytic review*. Neuropsychology, 2009. **23**(3): p. 315-36.

23. Savla, G.N., et al., *Deficits in domains of social cognition in schizophrenia: a meta-analysis of the empirical evidence*. Schizophr Bull, 2013. **39**(5): p. 979-992.
24. Schretlen, D.J. and A.M. Shapiro, *A quantitative review of the effects of traumatic brain injury on cognitive functioning*. Int Rev Psychiatry, 2003. **15**(4): p. 341-9.
25. Gillespie, D.C., A. Bowen, and J.K. Foster, *Memory impairment following right hemisphere stroke: a comparative meta-analytic and narrative review*. Clin Neuropsychol, 2006. **20**(1): p. 59-75.
26. Willcutt, E.G., et al., *Validity of the executive function theory of attention-deficit/hyperactivity disorder: a meta-analytic review*. Biological Psychiatry, 2005. **57**: p. 1336-1346.
27. Schmand, B., H.M. Huizenga, and W.A. van Gool, *Meta-analysis of CSF and MRI biomarkers for detecting preclinical Alzheimer's disease*. Psychol Med, 2010. **40**(1): p. 135-45.
28. Keefe, R.S., C.E. Eesley, and M.P. Poe, *Defining a cognitive function decrement in schizophrenia*. Biol Psychiatry, 2005. **57**(6): p. 688-691.
29. Seidman, L.J., et al., *Neuropsychological performance and family history in children at age 7 who develop adult schizophrenia or bipolar psychosis in the New England Family Studies*. Psychological Medicine, 2013. **43**(1): p. 119-131.
30. Gur, R.C., et al., *Neurocognitive growth charting in psychosis spectrum youths*. JAMA Psychiatry, 2014. **71**(4): p. 366-374.
31. Harvey, P.D. and R.S. Keefe, *Studies of cognitive change in patients with schizophrenia following novel antipsychotic treatment*. Am J Psychiatry, 2001. **158**(2): p. 176-84.
32. Mishara, A.L. and T.E. Goldberg, *A meta-analysis and critical review of the effects of conventional neuroleptic treatment on cognition in schizophrenia: opening a closed book*. Biol Psychiatry, 2004. **55**(10): p. 1013-22.
33. Thornton, A.E., et al., *The impact of atypical antipsychotic medications on long-term memory dysfunction in schizophrenia spectrum disorder: a quantitative review*. J Psychopharmacol, 2006. **20**(3): p. 335-46.
34. Keefe, R.S., et al., *Neurocognitive effects of antipsychotic medications in patients with chronic schizophrenia in the CATIE Trial*. Arch Gen Psychiatry, 2007. **64**(6): p. 633-47.

35. Knowles, E.E., A.S. David, and A. Reichenberg, *Processing speed deficits in schizophrenia: reexamining the evidence*. Am J Psychiatry, 2010. **167**(7): p. 828-35.
36. Reilly, J.L., et al., *Antipsychotic drugs exacerbate impairment on a working memory task in first-episode schizophrenia*. Biol Psychiatry, 2007. **62**(7): p. 818-21.
37. Beninger, R.J., et al., *Typical and atypical antipsychotic medications differentially affect two nondeclarative memory tasks in schizophrenic patients: a double dissociation*. Schizophrenia Research, 2003. **61**(2-3): p. 281-292.
38. McCleery, A., et al., *Cognitive impairment in first-episode schizophrenia: MATRICS Consensus Cognitive Battery (MCCB) Profile of Impairment*. Schizophrenia Research, 2014. **157**(1-3): p. 33-39.
39. Bilder, R.M., et al., *Neuropsychology of first-episode schizophrenia: initial characterization and clinical correlates*. 2000(0002-953X (Print)).
40. Seidman, L.J., et al., *Neuropsychology of the prodrome to psychosis in the NAPLS consortium: relationship to family history and conversion to psychosis*. Arch Gen Psychiatry, 2010. **67**(6): p. 578-88.
41. Bozikas, V.P. and C. Andreou, *Longitudinal studies of cognition in first episode psychosis: a systematic review of the literature*. Aust N Z J Psychiatry, 2011. **45**(2): p. 93-108.
42. Keefe, R.S. and W.S. Fenton, *How should DSM-V criteria for schizophrenia include cognitive impairment?* Schizophr Bull, 2007. **33**(4): p. 912-920.
43. Green, M.F., *What are the functional consequences of neurocognitive deficits in schizophrenia?* American Journal of Psychiatry, 1996. **153**(3): p. 321-330.
44. Green, M.F., R.S. Kern, and R.K. Heaton, *Longitudinal studies of cognition and functional outcome in schizophrenia: implications for MATRICS*. Schizophr Res, 2004. **72**(1): p. 41-51.
45. McGurk, S.R. and K.T. Mueser, *Cognitive functioning, symptoms, and work in supported employment: a review and heuristic model*. Schizophr Res, 2004. **70**(2-3): p. 147-73.

46. Niekawa, N., et al., *Relationship between financial competence and cognitive function in patients with schizophrenia*. Psychiatry and Clinical Neurosciences, 2007. **61**(5): p. 455-461.
47. Schutt, R.K., et al., *The role of neurocognition and social context in predicting community functioning among formerly homeless seriously mentally ill persons*. Schizophr Bull, 2007. **33**(6): p. 1388-1396.
48. Marwaha, S., et al., *Rates and correlates of employment in people with schizophrenia in the UK, France and Germany*. British Journal of Psychiatry, 2007. **191**(1): p. 30-37.
49. Lindamer, L.A., et al., *A comparison of gynecological variables and service use among older women with and without schizophrenia*. Psychiatric Services, 2003. **54**(6): p. 902-904.
50. Suslow, T., et al., *Prediction of work performance by clinical symptoms and cognitive skills in schizophrenic outpatients*. Journal of Nervous and Mental Disease, 2000. **188**(2): p. 116-118.
51. Fett, A.K., et al., *The relationship between neurocognition and social cognition with functional outcomes in schizophrenia: A meta-analysis*. Neurosci Biobehav Rev, 2011. **35**(3): p. 573-588.
52. Meyer, G.J., et al., *Psychological testing and psychological assessment. A review of evidence and issues*. The American Psychologist, 2001. **56**(2): p. 128-165.
53. Murray, C.J. and A.D. Lopez, *The Global Burden of Disease: A Comprehensive Assessment of Mortality and Disability from Diseases, Injuries and Risk Factors in 1990 and Projected to 2020*. 1996, Harvard School of Public Health, on behalf of the World Health Organisation and the World Bank, Cambridge, MA: Harvard University Press.
54. Keefe, R.S. and P.D. Harvey, *Cognitive impairment in schizophrenia*. Handbook of Experimental Pharmacology, 2012. **213**: p. 11-37.
55. Keefe, R.S., et al., *Efficacy and safety of donepezil in patients with schizophrenia or schizoaffective disorder: Significant placebo/practice effects in a 12-week, randomized, double-blind, placebo-controlled trial*. Neuropsychopharmacology, 2007. **33**(6): p. 1217-1228.

56. Wykes, T., et al., *Cognitive remediation therapy in schizophrenia: randomised controlled trial*. British Journal of Psychiatry, 2007. **190**: p. 421-427.
57. McGurk, S.R., et al., *A Meta-Analysis of Cognitive Remediation in Schizophrenia*. American Journal of Psychiatry, 2007. **164**(12): p. 1791-1802.
58. Kurtz, M.M., et al., *Approaches to Cognitive Remediation of Neuropsychological Deficits in Schizophrenia: A Review and Meta-Analysis*. Neuropsychology Review, 2001. **11**(4): p. 197-210.
59. McGurk, S.R., et al., *Cognitive training for supported employment: 2-3 year outcomes of a randomized controlled trial*. American Journal of Psychiatry, 2007. **164**(3): p. 437-441.
60. Wykes, T., et al., *The effects of neurocognitive remediation on executive processing in patients with schizophrenia*. Schizophr Bull, 1999. **25**(2): p. 291-307.
61. Kohen, J., *A coefficient of agreement for nominal scale*. Educ Psychol Meas, 1960. **20**: p. 37-46.
62. Landis, J.R. and G.G. Koch, *The measurement of observer agreement for categorical data*. Biometrics, 1977. **33**(1): p. 159-74.
63. Braun, V. and V. Clarke, *Using thematic analysis in psychology*. Qualitative research in psychology, 2006. **3**(2): p. 77-101.
64. Andrew, A., et al., *Effective interventions in schizophrenia: the economic case*. 2012.

APPENDIX 1: SCORING CRITERIA FOR STRUCTURED INTERVIEW SCHEDULE
QUESTION 1 (NAMING AND BRIEFLY DESCRIBING COGNITIVE IMPAIRMENTS IN
PSYCHOSIS AND SCHIZOPHRENIA)

Scoring Criteria for Naming/Briefly Describing Question

Participants are scored 0-9, based on how many of the following cognitive domains they identify *throughout the entirety* of the baseline interview, that is, not only in response to this (first) question, but also as part of responses to any other question throughout the interview. Participants only score once for each domain, regardless of how many times they may refer to that domain during the interview.

Participants receive a score, unless they go on to give a response that *clearly invalidates* this, e.g., if someone mentions difficulties in ‘responding appropriately’, which could potentially be scored due to being related to language problems, but then immediately goes on to discuss delusional thinking – which would then suggest that their response was in fact concerned with positive symptoms, rather than with cognitive impairment.

Cognitive impairments must be contextualized within the Schizophrenia diagnosis – and not explicitly linked by the participant to another diagnosis/es (e.g., Asperger’s syndrome, alcohol and/or other drug addictions, brain injury, etc.); cognitive impairments are assumed to be related to Schizophrenia in particular, unless the participant explicitly refers to another disorder (i.e. participants generally receive the benefit of the doubt).

Also, the participant must clearly and obviously identify the cognitive impairments that they may be describing as such – it is not enough for participants to merely mention cognitive impairments in passing without it being clear that they believe themselves to be referring to cognitive impairments. That said, participants do not have to actually name the domain that they are referencing (i.e. processing speed) in order to score, but they must, firstly, clearly describe such things as being a problem/difficulty, and secondly, describe the problem in such a way that clearly relates to that domain’s being the issue.

In general, the benefit of the doubt is given to participants in cases of uncertainty/ambiguity.

1. Attention/Concentration:

- Any clear reference to, or unambiguous examples of, difficulties in (paying) attention/concentration/focusing [on something] (e.g., difficulty concentrating when watching television, in conversation, being easily distracted, etc.)

2. Executive functions:

- Any clear reference, or unambiguous examples of, to one or more of the following abilities:
 - Planning/prioritizing (budgeting/finances)
 - Self-monitoring (spec. with reference to monitoring outcomes of behaviour, etc.)
 - Task initiation
 - Organisation
 - Working memory ('being unable to carry-out/process complex tasks, like making a cup of tea')
 - Sequencing
 - Problem-solving ('considering courses of action to take')
 - Inhibition/impulsivity
 - Decision-making
 - Cognitive flexibility

3. General intelligence:

- Any clear reference to, or unambiguous examples of, a lower general intelligence/IQ or 'general cognitive functioning'

The reference must be clearly related to a reduced IQ within the normal range, and not to learning disabilities (i.e. IQ < 70).

4. Language abilities:

- Any clear reference to, or unambiguous examples of, difficulties in language abilities (in verbal or written form)
- The reference must be clearly and specifically related to the understanding/comprehension or production of language in the context of

Schizophrenia, and *not to* either illiteracy or to English not being the person's native language

5. Memory:

- Any clear reference to, or unambiguous examples of, memory problems/difficulties remembering [information/something/things; i.e. semantic or episodic memory] or how to do something (procedural memory) (e.g., medication, appointments, conversations, things watched on television, use a mobile phone, etc.)

6. Motor abilities:

- Any clear reference to, or unambiguous examples of, difficulties in the production/execution/coordination of gross or fine muscle-movements

7. Processing-Speed:

- Any clear reference to, or unambiguous examples of, a longer period of time than normal being needed to process (any forms of) information (e.g., in conversation, when reading, etc.)

8. Social cognition:

- Any clear reference to, or unambiguous examples of, difficulties in the processing of social information and situations, as per the following:
 - Theory of Mind abilities
 - Social perception (understanding social roles, societal rules and social context)
 - Social knowledge (awareness of the rules and goals that govern and characterize social situations and interactions)
 - Emotional processing (the ability to use and perceive emotions)
 - The reference must not be as part of any comorbid neurodevelopmental disorders (e.g., autistic spectrum disorders) in which difficulties in social processing are key features

9. Visuospatial abilities:

- Any clear reference to, or unambiguous examples of, difficulties in the processing of visuospatial information (e.g., awareness of oneself in space, difficulties in map-reading, finding locations, etc.)

APPENDIX 2: SLIDES FROM TEACHING SESSION INTERVENTION DELIVERED TO MDT STAFF MEMBERS

COGNITIVE IMPAIRMENT IN PSYCHOSIS / SCHIZOPHRENIA

Dr. Matthew J. Mayhew
Clinical Psychologist in Training
Institute of Psychiatry, Psychology and Neuroscience (IoPPN)

Aims

- 1) Define 'cognition' and describe the main processes and abilities it incorporates
- 2) Describe the types of cognitive impairment often observed in those with Psychosis/Schizophrenia
- 3) Describe the effects on functioning of cognitive impairment in Psychosis/Schizophrenia
- 4) Common everyday manifestations of cognitive impairment
- 5) Everyday strategies to mitigate the effects of cognitive impairment

DEFINING AND DESCRIBING COGNITIVE IMPAIRMENT IN SCHIZOPHRENIA

Defining Cognition

- 'How the brain adapts to its environment in a way that helps the individual to fulfil their needs and achieve their goals' (Weiden, 2014)
- Incorporates a range of functions, including:
 - Processing speed → Ability to perform tasks quickly/efficiently
 - Attention/concentration → Ability to focus on something specific
 - Memory:
 - Short-term memory (STM) → Verbal STM and visual STM
 - Long-term memory (LTM)
 - Working memory → Holding and manipulating information
 - Speech and language abilities → Vocabulary, comprehension, expression
 - Visuo-spatial skills → Recognising, organising and interpreting visual information (e.g., map-reading)
 - IQ → Average performance across these abilities

Defining Cognition: Executive Functioning

- **Executive Functioning (EF):** umbrella term for a range of processes involved in the initiation and maintenance of goal-directed behaviour, including:
 - Planning/organisation
 - Initiation of action → 'Getting started' with something
 - Problem-solving
 - Impulse-control / Inhibition of inappropriate actions
 - Self-monitoring / regulation of ongoing actions
 - Set-shifting → Ability to 'flip back and forth' between tasks
 - Cognitive flexibility → Adaptability in response to changing conditions
- EF is associated with the prefrontal cortex



Defining Cognition: Social Cognition

- **Social Cognition:** 'The way in which people perceive, process and utilise social information' (Pintham, 2013, p. 126)
- Is crucial for adequate social functioning
- Incorporates a number of related skills/abilities:
 - The ability to identify social roles, societal rules, social context
 - Awareness of the rules and goals that characterise social situations
 - Understanding the intentions, emotions and dispositions of other people (as well as our own) → 'Theory of Mind' ability (Dennett, 1987)
 - Processing, interpreting and managing emotions in social situations

A Little History...

- Early conceptualisations of schizophrenia (Kraepelin [1896, 1919]; Bleuler [1943, 1950]) held a central role for cognitive impairments
 - Hence 'Dementia Praecox' ('premature cognitive decline')
- Cognitive impairment was considered 'fundamental', delusions and hallucinations 'accessory' (i.e. the reverse of current conceptualisations)
 - DSM (APA) and ICD (WHO) classification systems make no mention of cognitive impairments
 - Positive symptoms more noticeable
 - Possibly due to lack of standardised cognitive assessments
- Cognitive impairments are now receiving greater attention

Cognitive Impairment in Schizophrenia

- Those with Schizophrenia show impairments across all cognitive abilities (Heinrichs & Zakzanis, 1998)
- The very largest impairments are seen in:
 - Processing speed
 - Working memory
 - Attention
 - Executive functioning
 - Verbal memory
 - General intellectual ability
 - Social cognition
- But impairments are wide-ranging and also include:
 - Speech and language abilities
 - Visuo-spatial and motor abilities

Cognitive Impairment in Schizophrenia

- Those with Schizophrenia show impairments across all cognitive abilities (Heinrichs & Zakzanis, 1998)

75-98% of patients demonstrate cognitive deficits!

- But impairments are wide-ranging and also include:
 - Speech and language abilities
 - Visuo-spatial and motor abilities

Is Cognitive Impairment Brought About by Medication?

- **Overall:** No
 - A number of studies report anti-psychotic medications to have a mildly beneficial, rather than detrimental effect, on cognition in general (e.g., Harvey & Keefe, 2001; Mishara & Goldberg, 2004)
 - Similar findings replicated cross-culturally (e.g., in a Chinese sample; Guo et al., 2011)
- **However:**
 - **Slow processing speed** may be the result of, or at least augmented by, anti-psychotic medication (Knowles, David & Reichenberg, 2010)
 - **Aspects of memory** may also be impacted by medication (Beninger et al., 2003; Kelly et al., 2007)
- But overall, the evidence indicates that cognitive impairments are not caused by medication

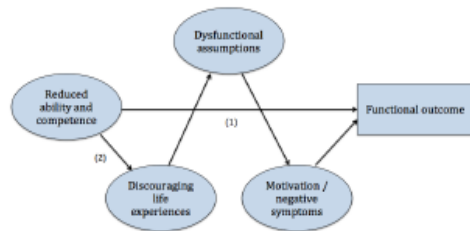
Is Cognitive Impairment Brought About by Stress?

- **This seems unlikely, as:**
 - 1) Moderate to severe impairment across most cognitive domains is already present in first-episode patients (Blaser et al., 2000; McCleery et al., 2004)...
 - 2) ... And milder impairment is observed in those exhibiting prodromal symptoms (Seldman et al., 2010)
 - 3) The deficit profile observed in first episode and prodromal individuals is consistent with that seen in more chronic samples
 - 4) The impairment observed in first-episode patients and prodromal individuals remains stable for up to 10 years (Bozilas & Andreou, 2011)

Cognitive Impairment and Functional Outcome

- **'Functional outcome'** includes:
 - Degree of independence in community living
 - Occupational development
 - Social relationships
 - Quality of life
- Cognitive impairment a stronger predictor of poor functional status (including unemployment) **THAN POSITIVE OR NEGATIVE SYMPTOMS** (Keefe & Fenton, 2007)
- Up to 23% of functional outcome is accounted for by aspects of cognition (Fett et al., 2011)
 - Mammogram results account for 9% of cancer mortality at 1-year follow-up (Meyer et al., 2011)

How Does Cognitive Impairment Affect Daily Functioning?



Interventions for Cognitive Impairment

- **Overall, medications seem to be of little benefit:**
 - Neither anti-psychotic medication nor pharmacological augmentation seem to produce improvements (Keefe & Harvey, 2012)
- **However, Cognitive Remediation Therapy (CRT) is effective**
 - An umbrella term referring to a range of different but related interventions, with the shared aim of improving impaired cognition
 - Often delivered by a therapist
 - The patient is given tasks to improve cognition in affected domains
 - Tasks are repeated over and over until the specific ability in question has improved
 - Medium to large improvements are seen across various domains of cognition (Kurtz et al., 2001; Wykes et al., 2011)
 - Patients report CRT to be engaging and enjoyable, associated with improved self-esteem (Wykes et al., 1999)

EVERYDAY MANIFESTATIONS OF COGNITIVE IMPAIRMENT

Impaired Attention: Everyday Manifestations

- **Everyday manifestations can include:**
 - Seeming confused or absent-minded
 - Seeming indifferent to the environment
 - Losing track of time
 - Difficulty concentrating or understanding things said or read
 - Difficulty maintaining a conversation
 - Interrupting others when they are talking
 - Difficulty remembering what they or others have said
 - Becoming distracted in the middle of doing things
 - Frequently commenting/complaining of boredom
 - Becoming easily overwhelmed
 - Wandering around in an 'aimless' manner

Impaired Memory: Everyday Manifestations

- **Everyday manifestations can include:**
 - Forgetting to take medication, names of / rationale for medication...
 - ... Or taking too much medication
 - Forgetting scheduled appointments
 - Having difficulty finding items around the house (e.g., keys, wallet)
 - Not following-through with plans (e.g., paying rent/utilities)
 - Losing track of money spent
 - Needing reminders about important dates (e.g., birthdays, holidays)
 - Repeating questions over and over again
 - Not being able to remember directions/instructions
 - Difficulty learning new information
 - Forgetting people's names
 - Difficulty remembering recent activities/current events
 - Having difficulty travelling around (e.g., to day-centres, etc.)

Impaired Speech and Language Abilities: Everyday Manifestations

- **Everyday manifestations can include:**
 - Taking longer than normal to respond to questions
 - Speech production slower
 - Not saying very much (e.g., 'yes/no' answers)
 - Difficulty maintaining a conversation / conversation break-down
 - Difficulty finding/remembering words
 - Difficulty remembering what's been said
 - Difficulty grasping the gist of verbal or written information
 - Taking longer than normal to read text
 - Difficulty understanding text
- Importantly, speech and language difficulties are also likely to result from slow processing speed
- All the common difficulties on these slides represent a combination of underlying difficulties, rather than being pure/simple effects of just one

Impaired Executive Functioning: Everyday Manifestations (1)

- **Everyday manifestations can include:**
 - Responding too quickly/impulsively
 - Seeming not to understand consequences of actions
 - Difficulty seeing one's own mistakes
 - Repeating mistakes without learning from previous errors (e.g., repeatedly over-spending, missing deadlines for paying bills)
 - Having trouble getting things started independently
 - Experiencing difficulty if/when routines are changed
 - Difficulty adjusting to new demands
 - Difficulty making decisions
 - Not seeming to plan ahead (e.g., spending benefits all at once, getting into debt)

Impaired Executive Functioning: Everyday Manifestations (2)

- Seeming indifferent to working-out practical problems
- Tending not to ask for help, even when experiencing difficulty
- Doing things in a disorderly or disorganised manner (e.g., living in an untidy, messy home)
- Difficulty multi-tasking (e.g., rarely cooking meals at home)
- Frequently not finishing things that have been started
- Appearing 'lazy' and poorly motivated to work things out
- Appearing rigid and 'concrete' / difficulty with abstract thought
- Over-sensitivity to rewarding stimuli (e.g., regularly eating rich, fatty foods, alcohol and other drug use/abuse, unprotected sex)
- Not evaluating that actions may be dangerous (e.g., alcohol and other drug use/abuse, smoking)
- Difficulty problem-solving and seeking-out alternative options (e.g., self-harm / suicide attempts)

Impaired Social Cognition: Everyday Manifestations

- **Everyday manifestations can include:**
 - Difficulty understanding the emotional states and intentions of others (e.g., frequent misunderstandings/arguments with others)
 - Difficulty adhering to 'unwritten' social rules
 - Frequently making faux-pas
 - Difficulty managing emotions in social situations
 - Aggressive behaviour
 - Inappropriate sexual statements / actions / advances
 - Tangential speech (e.g., 'going round the houses')...
 - ... Or not giving enough information sufficient for understanding
 - Social isolation / avoidance of social situations
 - Related to anxiety around / difficulty negotiating social situations
 - Vicious cycle

EVERYDAY STRATEGIES FOR MITIGATING COGNITIVE IMPAIRMENT

Any suggestions?

Everyday Strategies: Memory Difficulties

- Repeat information/instructions
- Regularly summarise:
 - Ask the person to repeat or paraphrase what you have just told them → do this frequently throughout a session
 - Repeat as necessary
 - Provide choices and cues to help them remember essential information
- Put things in writing (when possible):
 - Auditory information alone is problematic for those with poor memory
 - If the person writes down information, check this with them
- Use memory aids:
 - Calendars, diaries, dosette boxes, phone reminders and post-it notes are all useful tools to cue memory

Everyday Strategies: Attention Difficulties (1)

- Limit information to the span of attention:
 - Keep things simple, direct, short and to the point
- Don't expect someone to be able to do multiple tasks at the same time:
 - Divided attention is extremely difficult
- Direct eye contact can gain attention and sustain involvement
- Be aware of distractions (e.g., background noises, multiple speakers, poor acoustics) and attempt to simplify the environment

Everyday Strategies: Attention Difficulties (2)

- **Regulate tone, volume and rhythm of speech:**
 - Enthusiasm easily captures attention
- **The more personally involved an individual can become in a task, the greater their attention:**
 - It can be helpful to link tasks to personal goals (e.g., reminding the client to go to a day-centre in order to create new friendships, etc.)
- **Management of sessions**
 - Not too long, have breaks, conversational interludes

Everyday Strategies: Executive Functioning Difficulties (1)

- Encourage the use of routines, organisation and structure
- Encourage people to think about the potential advantages and disadvantages of engaging in reckless/unhealthy behaviour:
 - E.g., 'What are the positives and negatives about budgeting with finances?'
- Provide encouragement/praise for actions that are initiated or maintained and followed-through
 - For people who have difficulty getting started or tend not to complete tasks
- Offer guiding questions instead of ready-made answers for people who find it difficult to problem-solve:
 - E.g., 'What's the first step?', 'What do you think?'

Everyday Strategies: Executive Functioning Difficulties (2)

- Demonstrate procedures and sequences to develop everyday problem-solving
- Encouraging 'self-talk' (talking out loud):
 - This can help 'metacognition' – thinking about thinking – and connections between thoughts and actions
- Not making assumptions about whether/how a person can perform daily tasks without asking how they would solve the problem

Thank you for your time and for listening!

- Questions??
??
??
????????????????????????

APPENDIX 3: TYPES OF INTERVENTION STRATEGIES FOR COGNITIVE IMPAIRMENT IN SCHIZOPHRENIA MENTIONED BY PARTICIPANTS

Table A1 presents the types of intervention strategies for cognitive impairment in schizophrenia mentioned by participants, together with illustrative quotations and the numbers of participants that mentioned each strategy.

Table A1: Types of intervention strategies for cognitive impairment in schizophrenia mentioned by participants, as well as illustrative quotations and the numbers of participants that mentioned each strategy ($N = 15$)

Type of intervention (alphabetised)	Example of participant response	Number of participants that mentioned strategy
<i>Employment/engagement in meaningful activities</i>	‘I think it’s important that, erm, that they have some form of employment – I don’t mean paid employment, but some sort of activity that they involve themselves... that empowers them and provides stability’ (Participant 8)	4
<i>Encouraging/fostering independence</i>	‘... I try not to get into prompting somebody every time I’m seeing them, so that they can sort of get used to remembering appointments and checking their diary and, you know, taking responsibility for it, so...’ (Participant 12)	1
<i>Groups/socialising with others</i>	‘... We have a lot of groups that have been run here, to support people that are like... We have, erm, you know, the Walking	1

Table A1 continues over the page

Table A1 continued

Type of intervention (alphabetised)	Example of participant response	Number of participants that mentioned strategy
	Group, we have the Women [sic] Group, we have, erm, Bipolar Group, to support people at that level to do that... A lot of things are done to support them' (Participant 13)	
<i>Improving sleep hygiene</i>	'... There's probably medication, as well, that could help, erm, if someone's sleep's impaired... that could help promote better concentration during the day, erm, and any sleep hygiene interventions that are not medication-related' (Participant 7)	1
<i>Managing anxiety (and thereby improving impairments indirectly)</i>	'... I would see that much more as, not managing cognitive impairments, but managing anxiety or managing, you know, helping someone to-, I don't see it in terms of cognitive impairment...' (Participant 14)	1
<i>Medication:</i>		
<i>To act directly on impairments</i>	'... Maybe medication also would be... an intervention that might help...' (Participant 4)	3
<i>Managing the side-effects of medication</i>	'... The side-effects of medication is [sic] talked about <i>a lot</i> ... Maybe looking at the side-effects of medication, as that [sic]	2

Table A1 continues over the page

Table A1 continued

Type of intervention (alphabetised)	Example of participant response	Number of participants that mentioned strategy
	<i>could</i> be something that's creating the problem...' (Participant 11)	
<i>Not knowing whether/which interventions might help</i>	Interviewer: Okay, and do you know anything about what can be done to manage cognitive impairments in psychosis and schizophrenia? Participant 9: Not really... No, if I'm honest, no.	3
<i>Occupational therapy-related / practical interventions and adaptations (e.g., diaries, phone reminders)</i>	'... It's about breaking-down, erm, information, breaking-down everyday barriers for people... helping people to keep things, like diaries, that works for them or, erm, alarms...' (Participant 1)	10
<i>Otherwise unspecified form of therapy</i>	'Therapy might help...' (Participant 3)	1
<i>Psychology-related approaches: 'Behavioural' approaches</i>	'... I would've thought that... something, maybe, along the sort	1

Table A1 continues over the page

Table A1 continued

Type of intervention (alphabetised)	Example of participant response	Number of participants that mentioned strategy
	of behavioural therapy lines might work, with a specific, erm, impairment, so maybe, er, actually, retraining somebody, for instance, take an example of how, how, how do you, you know, if somebody's coming the other way on a towpath, or whatever, erm, how to recognise that and maybe take avoiding action, I guess you could do a behavioural thing around that, you know, actually get people to perform that in a sort of, erm, er, you know, a role-play...' (Participant 4)	
<i>Cognitive-Behavioural Therapy (CBT)</i>	'... Well, I think the work we do... CBT-kind of approaches that help people to start to challenge... these beliefs...' (Participant 10)	2
<i>Cognitive Remediation Therapy (CRT)</i>	'For a while, there was this Cognitive Remediation Therapy...' (Participant 2)	2
<i>Collaborative, person-centred approaches</i>	'... Well, it depends on the cognitive impairment, so it's looking at how one balances the person's strengths with the person's weaknesses... and saying, "... Let's look at how we can	4

Table A1 continues over the page

Table A1 continued

Type of intervention (alphabetised)	Example of participant response	Number of participants that mentioned strategy
<i>Mindfulness</i>	support you and you can support yourself in the areas that, er, that there are these deficits.” (Participant 5)	
	‘... Mindfulness-type things might help to focus the mind, erm, keep them... grounded (Participant 7)	2
<i>Psychoeducation</i>	‘... You know, with cognitive deficits, it’s, it’s often where, where a person <i>feels</i> there’s something not going well for them, you know, so it’s not us telling them, “You’ve got a cognitive deficit” and they’re completely unaware of it; it’s more someone that’s, feels that they’re not getting on with something, something’s stopping them moving-on in life in one direction, and helping them better understand it – that it’s a cognitive deficit, it’s not because they’re, you know, stupid or lazy or some of those other ways that people will take these on themselves...’ (Participant 5)	2
<i>Support worker</i>	‘... Having a support person, erm... support people with their difficulties’ (Participant 7)	2

Table A1 continues over the page

Table A1 continued

Type of intervention (alphabetised)	Example of participant response	Number of participants that mentioned strategy
<i>Treating the (positive) symptoms of schizophrenia (and thereby improving impairments indirectly):</i>		
<i>Via medication</i>	‘Number one, I think, managing the illness, so that there aren’t episodes of, erm, acute <i>illness</i> , which cause ongoing damage to your white matter and neurones... medication would be one part of it...’ (Participant 8)	1
<i>Via increasing insight</i>	‘... We can speak to them and try to explain to them that what they’re speaking about is maybe not really what is going on...’ (Participant 6)	3

CLINICAL CASE STUDY 1

A PROBLEM IN SPECIFYING THE PROBLEM:
COMPLICATIONS IN A
COGNITIVE/NEUROPSYCHOLOGICAL MEMORY
ASSESSMENT OF A PATIENT WITH
PSYCHOSIS/SCHIZOPHRENIA

SUPERVISED BY DR. ABIGAIL CLARK

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I. LITERATURE REVIEW

I.1) DEMENTIA

‘Dementia’ is an umbrella term referring to a range of degenerating brain conditions, all of which involve the progressive deterioration of cognitive functions – their common feature being a decline in memory functioning [1]. The two most common forms of dementia are Alzheimer’s disease (AD) and vascular dementia (VD) [2]. AD is characterised by the development of neurofibrillary tangles (groupings of protein within neurons), senile plaques (accumulations of protein between neurons) and cortical atrophy in a number of brain regions [3]. Initially, these neural abnormalities tend to be concentrated within regions of the medial temporal lobe associated with episodic memory (EM) – a form of long-term memory concerned with personal experience [4]; consequently, EM tends to be the first aspect of cognition to deteriorate [5]. As the underlying neuropathology progressively expands to other brain regions, so the EM impairment becomes progressively worse, along with deteriorations in executive functioning [functions comprising volitional activities, such as planning, organising, self-awareness, self-regulation and the initiation of action; 6], semantic memory [a form of long-term memory concerning general knowledge about the world, language, etc.; 4] and attention and visuo-spatial abilities [7, 8]. Initially, these difficulties may only be apparent to the individual but, with time, they become increasingly apparent to others. In the advanced stages of the disease, the sufferer is unable to live independently and may even be ignorant of their cognitive impairment(s). In contrast to the gradual onset of AD, VD occurs abruptly, in the wake of infarction(s). Severer forms of VD tend to result from pronounced infarcts, which leave a region of dead (or dying) brain tissue, whereas milder forms result from partial infarction within capillaries [9]. Although there is considerable overlap in the cognitive signs of AD and VD, VD patients typically exhibit greater executive impairments but fewer and less severe impairments in memory [10]. AD and VD can coexist in the form of ‘mixed dementia’, although the prevalence rates of this disorder are controversial [11].

Early-onset dementia (EOD) refers to Dementia occurring prior to 65 years of age; late-onset dementia occurs after 65 [12]. In terms of prevalence, EOD is experienced by around 0.1 per cent of people between the ages of 40 and 65. It is estimated that around

18,500 people in the UK suffer from EOD [13]. Beyond 65 years of age, incidence rates double approximately every five years, such that, by 85 years, approximately 50 per cent of people are estimated to have AD and other dementias [14].

I.II) PSYCHOSIS/SCHIZOPHRENIA

The term 'schizophrenia' refers to a heterogeneous range of symptoms, including implausible and peculiar beliefs and sensory experiences, social withdrawal, restricted or inappropriate emotional expression and disorganised behaviour [1]. Hallucinations and delusions have traditionally been considered as the core features of the disorder [15]. Prognoses are variable, with approximately one third of individuals experiencing just one episode in their lifetime, one third experiencing intermittent episodes interspersed with periods of remission and another third experiencing a chronic, deteriorating course [16]. The lifetime individual risk for developing schizophrenia is approximately 1 per cent and, on average, around 20 new cases per 100,000 are diagnosed each year [17].

I.III) COGNITIVE/NEUROPSYCHOLOGICAL DEFICITS IN PSYCHOSIS/SCHIZOPHRENIA

A number of meta-analytic studies report approximately 70-75% of individuals with schizophrenia to perform below general population norms for many standard cognitive tasks [15]. The magnitude of such differences is in the order of 1.5 pooled standard deviations for processing speed and aspects of sensory, verbal and working memory; differences of 1.0 standard deviation have been reported across tests of attention, executive functioning, language, motor and spatial abilities, as well as general intelligence [18-22]. To put this into perspective, the magnitude of these differences in cognition equal or exceed effect-sizes for moderate to severe traumatic brain injury and composite measures of cognition [23], the effects of right cerebral hemisphere stroke and non-verbal memory [24] and preclinical and subsequent Alzheimer's disease and memory performance [25]. The functional consequences of cognitive impairments appear to be significant, with degree of independence in community living, skill-acquisition, work performance and occupational development, social skills and relationships, as well as quality of life, all being negatively associated with neuropsychological deficits [26, 27].

Though it seems that the breadth and magnitude of cognitive impairments in schizophrenia cannot be attributed to anti-psychotic medication [28-30], highly-medicated patients demonstrate specific impairments in processing-speed, relative to less medicated patients [31]. There is also evidence that risperidone may disrupt spatial working memory [32].

I.IV) SCHIZOPHRENIA AND DEPRESSION AND ANXIETY DISORDERS: PREVALENCE AND EFFECTS ON COGNITION

Depression and anxiety disorders are commonly observed throughout the course of psychosis, including the prodromal phase and following symptomatic recovery [33-35]. Rates of comorbid depression have been estimated at between 30 and 50% [36, 37] and rates of anxiety disorders are thought to be around 45% [34]. The nature and course of these co-morbidities in schizophrenia are not well understood [38, 39]. Regardless, the presence of depression and anxiety disorders are both related to memory problems and other difficulties in cognition that can resemble those observed in dementia. Depression is associated with difficulties in EM, as well as broad impairments in executive functioning [40, 41], and anxiety disorders are associated with impairments in attention, executive functioning, long- and short-term verbal and visual memory and poorer processing speed [42]. In addition, depression is also highly co-morbid in dementia [43]. Depression and anxiety disorders are therefore important differential diagnoses when testing for dementia.

II. PRESENTING PROBLEM

The patient was a 58-year-old white British male outpatient with a primary diagnosis of psychosis/schizophrenia. His clinical psychologist had referred him for a formal memory assessment, following the patient's self-reporting long-standing memory problems, which seemed to be interfering with his daily functioning. In particular, the patient described forgetting appointments and everyday tasks, such as taking his medication. Since he had been prescribed the anti-psychotic Risperidone (6 mg daily), as well as insulin for diabetes, there were potentially serious consequences of his forgetting to take his medication. In addition, the patient reported difficulty following the conversation of his partner when at home and an inability to recall conversations that had occurred just half an hour previously. He also described often forgetting the purpose for which he has entered a room. The patient reported no difficulties, however,

in recalling events from his childhood and young adulthood (the accuracy of which were verified by his long-term partner). Thus, there were concerns that he may be exhibiting signs of early-onset dementia.

III. BACKGROUND

The patient had an approximately 20-year psychiatric history. Although he had begun to hear voices in the mid-1990s, the patient remained unknown to mental health services until December 2000, when he experienced an acute psychotic episode and was admitted to hospital as an inpatient.

He has experienced chronic low mood, marked by lack of pleasure and poor motivation. In 2004, he scored 43 on the Beck Depression Inventory, placing him in the severely depressed range. There was also a history of chronic anxiety, with a substantial social element. In social occasions, for example, he reports finding it hard to speak and is subsequently critical of himself. A previous therapist, working with the patient around low mood, poor motivation and self-critical thoughts, reported that he was, 'Too anxious to do the necessary exploratory work'. Such was his anxiety that she was unable to derive a formulation from the limited account of his difficulties that the patient was able to give. Similarly, another therapist reported that, '... He could respond to direct questioning but his answers were brief and unelaborate... He reported experiencing discomfort when questioned.'

In April 2000, the patient was involved in a road traffic accident, in which he was hit by a van and knocked from his bike. During a previous neuropsychological assessment, he reported losing consciousness for around 30 seconds, but remembered lying in the road and being helped-up after the accident. He sustained a cut on the back of his head and was taken to hospital via ambulance, where the wound was stitched, before he was discharged. Although the patient required a couple of weeks off work, he did not note any changes in his memory or concentration following the accident. A subsequent discharge summary reported no evidence of neurological deficits. He had been unemployed since this accident, but reports his giving-up work to be related to his psychiatric state, rather than any possible changes that may have occurred subsequent to the accident.

The patient is highly supported by his partner, who performs most activities of daily living (ADLs; daily self-care activities, e.g., bathing, dressing, grooming, etc.) for him.

This notwithstanding, and according to patient and partner report, when his partner is not present, the patient is capable of completing ADLs independently.

IV. PREVIOUS ASSESSMENT

Due to the patient having had concerns about his memory as far back as 2000, he previously underwent an in-depth assessment in 2005 (i.e. following his bicycle accident). The assessment comprised an interview with the patient, as well as completion of: (i) the Wechsler Adult Intelligence Scale-UK Third Edition [WAIS-IIIUK; 44], to assess his cognitive functioning; (ii) the National Adult Reading Test [NART; 45] and the Wechsler Test of Adult Reading [WTARUK; 46], to estimate his premorbid level of intellectual functioning; (iii) to assess his memory functioning, four subtests from the Adult Memory and Information-Processing Battery [AMIPB; 47] and the Short Recognition Memory for Words and Faces, from the Camden Test Battery [48]; and (iv) to assess his executive functioning, the Trail-Making Test [49], the Hayling Sentence-Completion Test [50], the Stroop task [51] and the Brixton Spatial Anticipation Test [50].

The following is a summary of the patient's results (see Appendix 1 for a detailed overview of his performance). In terms of his general intellectual ability, as assessed by the WAIS-III^{UK}, the patient's functioning was in the Low Average to Borderline ranges – which represented a significant level of under-functioning, relative to his premorbid, reading-based estimates derived from the WTAR and NART. His Performance IQ was particularly poor, which was attributed to slow processing speed. Regarding his memory, the patient performed either in the Below Average or Well Below Average ranges across tests, with his delayed recall seeming to be particularly impaired. Notably, however, the patient's forced-choice recognition of faces was at ceiling level and he made no errors on this task. In terms of the patient's executive functioning, there were marked difficulties with response-inhibition, sequence-detection and rule-following. Overall, based on the interview with the patient and his performance across the tests, it was concluded that it was, '... Difficult to attribute the current executive function difficulties, along with his slowed processing speed and memory difficulties, to [his bicycle accident in 2000] and it may be more likely that his psychiatric history and its treatment account for his current cognitive profile.'

V. AIMS OF THE PRESENT ASSESSMENT

The aim of the present assessment was to formally examine the patient's memory functioning, in order to i) establish whether any impairment/s were evident and ii) if so, to examine the extent to which the problem/s were organic, functional or both and iii) to inform his ongoing care plan with the community mental health team. Also, the results of the present assessment could be used as a baseline against which to compare the results of potential future assessments, in order to investigate the presence and progression of potential dementing conditions. The patient was keen to understand, 'What is going on?'

It was not possible, due to a lack of access, to administer the same tests as those given in the patient's previous neuropsychological assessment. The results of the previous assessment were nevertheless informative: the overall impression they gave of the patient's functioning in 2005 were compared to the present results, in order to explore whether his memory problems might have markedly worsened since then; if so, this would indicate a possible dementia.

VI. ASSESSMENT MEASURES

The following measures were administered (see Appendix 2 for detailed descriptions of each of these indices): (i) The Spielberger State and Trait Anxiety Inventory [STAI; 52]; (ii) The Test of Premorbid Functioning – UK Edition [TOPFUK; 53]; (iii) The Wechsler Memory Scale – UK 4th Edition IV [WMS-IVUK; 54]; (iv) The 2-Subtest Version of the Wechsler Abbreviated Intelligence Scale – UK 2nd Edition [WASI-IIUK; 55], and; (v) The Wisconsin Card-Sorting Test [WCST; 56].

VII. ASSESSMENT PROCEDURE

The assessment took place over two sessions; the first lasted around 1 hour and 20 minutes, whilst the second lasted around 2 hours and 15 minutes (see Table A7 in Appendix 3 for schematic over of measures administered and their sequencing/timings). The first session began with a brief interview concerning the patient's perspective of his memory difficulties. This was also used as a means of easing the patient into the assessment, with the aim that he did not begin the first test of the battery at peak anxiety. (I also tried, via my manner and tone of voice, to ease the patient's anxiety throughout the two sessions.) Following this, the STAI, TOPF^{UK} and

the first half of the WMS-IV^{UK} (i.e. subtests 1-5) were completed. The second half of the WMS-IV^{UK} (i.e. subtests 6-10), the WASI-II^{UK} and the WCST were completed in the second session.

VIII. ASSESSMENT RESULTS

VIII.I) BEHAVIOUR DURING THE ASSESSMENT

The examinee engaged well with the assessment and informed me that he was keen to participate. He seemed to understand the instructions of the tasks and what was required of him in each. However, he spent little time planning for tasks and did not appear to be implementing strategies in order to successfully complete the tasks. Similarly, in those tasks providing corrective feedback, he did not appear to learn from feedback indicating that he had given an incorrect answer, instead perseverating with an incorrect approach to the task. At the beginning of tasks, the patient would often state, 'I don't know' or 'I can't remember' and, despite encouragement, would not spend much time trying to problem-solve, instead giving up. With support from the examiner, however, he was able to complete tasks to a slightly higher level than recorded in this assessment. This may have been because he was being reminded of the instructions of the task, his focus was being kept on the task and/or he was encouraged to persevere.

VIII.II) RESULTS ON MEASURES

The patient's results for the various measures, described only briefly below, are related in more detail in Appendix 4.

VIII.II.I) THE STAI

The patient's state anxiety (s-anxiety) was 67 and his trait anxiety (t-anxiety) was 75, indicating considerable anxiety.

VIII.II.II) THE WMS-IV^{UK}

The patient's performance on the WMS-IV^{UK} indicated substantial and global memory impairment. The examinee's performance across 4 indices (auditory memory, visual memory, immediate memory and delayed memory) of the WMS-IV^{UK} fell into the extremely low range; his visual working memory performance fell into the borderline range.

VIII.II.III) THE TOPF^{UK}

The patient's TOPF^{UK} raw score was 56, placing his premorbid estimates in the average to high average range. His estimated premorbid scores for WMS-IV^{UK} indices were uniformly substantially greater than his obtained scores.

VIII.II.IV) THE WASI-II^{UK}

The examinee's performance on the WASI-II^{UK} placed him within the borderline range for FSIQ.

VIII.II.V) THE WCST

The patient's performance on the WCST demonstrated that his executive functioning was significantly impaired.

IX. DISCUSSION

The patient's performance across the WMS-IV^{UK}, TOPF^{UK}, WASI-II^{UK} and WCST was suggestive of significant memory, cognitive and executive impairments across all domains. Based on the examinee's face-value performance across all tasks in the current assessment, therefore, he would appear to be severely compromised, both in terms of his memory and his broader cognitive functioning.

There can be little doubt, based on the patient's self-report on his everyday experience and his significantly compromised WMS-IV^{UK} performance, that he is exhibiting memory dysfunction. This is consistent with literature suggesting difficulties with aspects of memory in those with psychosis/schizophrenia [15]. However, it is difficult, if not impossible, to pinpoint the extent to which this is the case. It is possible that the patient's impaired memory performance may have partly resulted from possible executive functioning difficulties, as suggested by his WCST performance – such

problems being common amongst those with schizophrenia/psychosis [20, 21]. Individuals with executive dysfunction are likely to perform poorly on a wide range of tasks/activities, memory or otherwise, due to so many relying, to some extent, on executive abilities [57]. This would be consistent with the examinee's impaired performance across all the measures administered. Nevertheless, it is not possible, on this assessment alone, to disentangle the patient's poor memory performance from his poor executive performance.

It is also possible that a third factor – the patient's apparently high level of state anxiety – may have significantly contributed to his impaired performance, raising a significant question-mark against the validity of the present assessment. (Questions concerning the validity of the assessment were also raised by the statistically-improbable TOPF^{UK} findings and the questionable WASI-II^{UK} results.) When asked during the sessions how he felt, the examinee verbally reported that he did not feel particularly anxious or worried – with which his body language and presentation during these sessions appeared consistent. However, there are several reasons for hypothesising that the examinee was in fact likely to have been experiencing significant anxiety. Firstly, it was well-documented in his notes that he experiences clinically-significant anxiety, and particularly in social situations. Since social anxiety is known to be related to a fear of negative evaluation by others [58], it is reasonable to conclude that a somewhat formal neuropsychological testing scenario, administered by someone the patient had not previously met, would have evoked a strong anxious response. Secondly, he self-reported a considerable level of state anxiety via the STAI. Thirdly, the patient did not present on the initial date set for the first assessment session due to fears that he had developed concerning the nature of the sessions: specifically, he had come to believe that he was to undergo brain surgery. This had understandably distressed him and appears to have led to his experiencing a protective, fugue-like state; thus, after having left his house to present for the first assessment session, the patient appeared to have forgotten the appointment and had instead spent hours walking around his local area. A similar incident occurred on what had initially been set as the second session. Following both of these occasions, he did, however, attend a rescheduled appointment for the following week. (Following his non-attendance of both of these appointments, the examiner checked whether the patient still wished to proceed with the assessment, to which he responded affirmatively on both occasions.) Incidentally, reports in his notes document that this fugue-like state occurs in other potentially anxiety-provoking

(social) situations, such as when meeting for the first time with a member of his care team.

Consequently, the patient's performance in the current assessment may significantly underestimate his 'true' memory and cognitive abilities. Indeed, were his performance in the present assessment to be truly representative of his ability, it would be virtually impossible for him to maintain the independent life as an outpatient that he does. Rather, if he was indeed experiencing the high level of anxiety hypothesised, it is reasonable to suggest that he was unable to focus his attention upon the requirements of the various tasks and to utilise his cognitive resources to his fullest ability level. His uniformly impaired performance across all tasks is consistent with this [59]. Furthermore, the patient's high trait anxiety score indicates that his high state anxiety was likely to be consistent across situations, and not specific to the assessment; it therefore follows that any memory problems related to such anxiety may *also* be consistent across situations in his everyday life.

It is also possible that the patient's chronic low mood (as documented in his care record) may also have impacted on his performance, depression being associated with impairments in memory and executive functioning, in particular [60]. Unfortunately, a measure of depression was not administered as part of the current assessment (a limitation), meaning that this remains unclear.

In the patient's previous assessment (in 2005), his memory performance, across all tests, fell into either the below average or well below average ranges, with his delayed recall particularly poor. The patient made no errors on a forced-choice recognition task. As detailed above, there was no such differentiation in the patient's scores in the present assessment, his performance across all indices falling into the extremely low range. It is therefore possible that the patient's memory functioning has indeed deteriorated since his previous assessment. Correspondingly, his general cognitive functioning in the previous assessment was in the low average to borderline range, whereas his performance here placed him in the borderline range. However, whereas the previous assessment used the full WAIS-III^{UK}, the present assessment administered the 2-subtest version subtest of the WASI-II^{UK} – a less reliable indicator of functioning. It is similarly difficult to compare the patient's memory and executive performances across the two assessments, given the different measures used to index these constructs in each assessment. Overall, then, it is not possible to directly compare performance

across the two assessments, meaning that a possible decline (i.e. owing to a dementing condition) in the patient's cognitive functioning, though possible, cannot be determined.

In conclusion, the true extent of the patient's memory dysfunction is unclear. It is possible that he has experienced memory, executive and more general cognitive deterioration as a result of his long-standing experiences of psychosis/schizophrenia [40, 41]. It may be that his taking risperidone may attenuate these difficulties [18-22]. It also seems likely that his (chronic) anxiety (and low mood) significantly exacerbates these difficulties by reducing his ability to concentrate and to fully devote his available cognitive resources to ongoing tasks and activities. It is, of course, also possible that the patient's performance is indicative of an underlying, organic memory dysfunction (i.e. dementia); yet it is not possible to discern this at the present stage.

IX.1) RECOMMENDATIONS

In terms of recommendations, ideally, I would suggest that the patient continue to receive psychological input for his anxiety, with a view to his repeating the WMS-IV^{UK} in the near future. A possibly reduced level of performance anxiety in a subsequent assessment may provide a clearer picture of the extent of his memory impairment. Unfortunately, he is soon to be discharged by his service; and besides, his performance anxiety in the current assessment followed significant anxiety work as part of his (substantial) course of psychological therapy. It is therefore unclear to what extent his performance anxiety may be improved in preparation for another assessment. Indeed, the potential benefits of his undergoing a further assessment, against the anxiety that this is likely to again engender, are debatable. Instead, it is recommended that the patient be referred for a course of cognitive remediation therapy, in order that he can learn and consolidate strategies to better manage his memory difficulties in everyday life.

IX.II) WIDER IMPLICATIONS

The questions concerning the validity of the present assessment raise questions more widely, concerning the validity of cognitive/neuropsychological findings in psychosis/schizophrenia. A large, and growing, literature documents significant cognitive impairment in those with schizophrenia, relative to non-psychiatric individuals [32]. Rates of anxiety disorders amongst those with psychosis are estimated to be around 45% [61]. However, the majority of studies examining cognitive impairments in schizophrenia do not seem to screen for the presence of anxiety disorders in their participants. It is therefore intriguing to speculate on whether the impaired performance of schizophrenic participants may be influenced, at least in part, by a proportion of individuals in these studies experiencing substantial levels of (performance) anxiety; that is, these studies may be underestimating the ‘true’, ‘underlying’ cognitive abilities of those with psychosis. It may therefore be advisable for future studies to measure, and statistically control for, comorbid anxiety, so as to examine whether/the extent to which anxiety might impair performance on cognitive/neuropsychological assessments. A potential future benefit of such procedures may be the generation of an algorithm to estimate the extent to which performance on a given individual neuropsychological assessment might have been adversely affected by performance anxiety. Alternatively, studies investigating cognitive deficits in psychosis may seek to exclude those who breach cut-off for clinically significant levels of anxiety, in the pursuit of a ‘purer’ estimate of cognitive impairment. However, such studies may be open to the accusation of being somewhat unrepresentative, given the relatively high proportions of anxiety disorders in those with schizophrenia.

X. REFLECTIONS

Throughout the assessment sessions, I noticed that I felt somewhat uneasy – guilty, even. I believe this could be traced to at least four inter-related thoughts: firstly, the examinee’s performance being quite obviously impaired across all tasks; secondly, his being very likeable and endearing, leading to feelings of warmth and sympathy towards him; thirdly, my being the facilitator of what appeared to me to be a rather unpleasant experience for the examinee; and finally, the manualised nature of neuropsychological assessment dictating that I was unable to provide any help or hints to the examinee, despite his obviously struggling to complete the tasks successfully. Related to the last

point, I was also aware of my experiencing feelings of frustration and tension throughout: observing somebody struggling to correctly perform a task, whilst resolutely failing to provide them with any tangible help is a deeply unpleasant, unusual, and arguably 'inhuman', experience: consequently, I felt somewhat relieved on those occasions when the examinee violated a discontinue criterion, meaning that that particular task could be ended, offering a brief respite until the next was started. These feelings notwithstanding, I have a strong interest in neuropsychology and would like to pursue a career in this area. I believe, therefore, that the present assessment will prove to be beneficial, in terms of my learning to notice and accept such difficult feelings. Although it may feel unpleasant watching someone struggle to perform in a cognitive/neuropsychological assessment, the primary purpose of such assessments is precisely this: to detect potential difficulties, in order that they can, hopefully, subsequently be ameliorated or remediated and the individual's functioning and quality of life improved.

REFERENCES

1. Association, A.P., *Diagnostic and Statistical Manual of Mental Disorders: DSM-5 (5th Ed.)*. 2013, Washington, D.C.: American Psychiatric Association.
2. Desmond, D.W., *Vascular dementia: a construct in evolution*. *Cerebrovasc Brain Metab Rev*, 1996. **8**(4): p. 296-325.
3. Lantos, P.L. and N.J. Cairns, *The neuropathology of Alzheimer's Disease*, in *Dementia*, J.O. O'Brien, D. Ames, and A. Burns, Editors. 2000, Arnold: London. p. 443-459.
4. Eysenck, M.W. and M. Keane, *Cognitive Psychology: A Student's Handbook*. 4th ed. 2000, Hove, East Sussex: Psychology Press, Ltd.
5. Hannay, H.J., et al., *Neuropathology for Neuropsychologists*, in *Neuropsychological Assessment (4th Ed.)*, M.D. Lezak, Editor. 2004, Oxford University Press: Oxford. p. 157-285.
6. Reber, A.S. and E.S. Reber, *Dictionary of Psychology*. 3rd ed. 2001, Strand, London: Penguin Group.
7. Butters, N., et al., *Differentiation of amnesic and demented patients with the Wechsler Memory Scale-Revised*. *Clinical Neuropsychologist*, 1988. **2**(2): p. 133-148.

8. Hodges, J.R. and K. Patterson, *Is semantic memory consistently impaired early in the course of Alzheimer's disease? Neuroanatomical and diagnostic implications*. Neuropsychologia, 1995. **33**(4): p. 441-59.
9. Mielke, R., et al., *Severity of vascular dementia is related to volume of metabolically impaired tissue*. Arch Neurol, 1992. **49**(9): p. 909-13.
10. Looi, J.C. and P.S. Sachdev, *Differentiation of vascular dementia from AD on neuropsychological tests*. Neurology, 1999. **53**(4): p. 670-8.
11. Zekry, D., J.J. Hauw, and G. Gold, *Mixed dementia: epidemiology, diagnosis, and treatment*. J Am Geriatr Soc, 2002. **50**(8): p. 1431-8.
12. Miyoshi, K., *What is 'early onset dementia'?* Psychogeriatrics, 2009. **9**(2): p. 67-72.
13. Harvey, R.J., *Young Onset Dementia: Epidemiology, Clinical Symptoms, Family Burden, Support and Income*. 1998, London: Dementia Research Group.
14. Bachman, D.L., et al., *Incidence of dementia and probable Alzheimer's disease in a general population: the Framingham Study*. Neurology, 1993. **43**(3 Pt 1): p. 515-9.
15. Heinrichs, R.W., et al., *Cognition as a central illness feature in schizophrenia*, in *Cognitive Impairment in Schizophrenia: Characteristics, Assessment and Treatment*, P.D. Harvey, Editor. 2013, Cambridge University Press: The Edinburgh Building, Cambridge.
16. Bentall, R.P., *Madness Explained: Psychosis and Human Nature*. 2003, London: Penguin Books.
17. Birchwood, M. and C. Jackson, *Schizophrenia*. Clinical Psychology: A Modular Course. 2001, Hove, East Sussex: Psychology Press, Ltd.
18. Mesholam-Gately, R.I., et al., *Neurocognition in first-episode schizophrenia: a meta-analytic review*. Neuropsychology, 2009. **23**(3): p. 315-36.
19. Johnson-Selfridge, M. and C. Zalewski, *Moderator variables of executive functioning in schizophrenia: meta-analytic findings*. Schizophr Bull, 2001. **27**(2): p. 305-16.
20. Aleman, A., et al., *Memory impairment in schizophrenia: a meta-analysis*. Am J Psychiatry, 1999. **156**(9): p. 1358-66.

21. Forbes, N.F., et al., *Working memory in schizophrenia: a meta-analysis*. Psychol Med, 2009. **39**(6): p. 889-905.
22. Heinrichs, R.W. and K.K. Zakzanis, *Neurocognitive deficit in schizophrenia: a quantitative review of the evidence*. Neuropsychology, 1998. **12**(3): p. 426-45.
23. Schretlen, D.J. and A.M. Shapiro, *A quantitative review of the effects of traumatic brain injury on cognitive functioning*. Int Rev Psychiatry, 2003. **15**(4): p. 341-9.
24. Gillespie, D.C., A. Bowen, and J.K. Foster, *Memory impairment following right hemisphere stroke: a comparative meta-analytic and narrative review*. Clin Neuropsychol, 2006. **20**(1): p. 59-75.
25. Schmand, B., H.M. Huizenga, and W.A. van Gool, *Meta-analysis of CSF and MRI biomarkers for detecting preclinical Alzheimer's disease*. Psychol Med, 2010. **40**(1): p. 135-45.
26. Green, M.F., et al., *Neurocognitive deficits and functional outcome in schizophrenia: are we measuring the "right stuff"?* Schizophr Bull, 2000. **26**(1): p. 119-36.
27. Green, M.F., R.S. Kern, and R.K. Heaton, *Longitudinal studies of cognition and functional outcome in schizophrenia: implications for MATRICS*. Schizophr Res, 2004. **72**(1): p. 41-51.
28. Harvey, P.D. and R.S. Keefe, *Studies of cognitive change in patients with schizophrenia following novel antipsychotic treatment*. Am J Psychiatry, 2001. **158**(2): p. 176-84.
29. Keefe, R.S., et al., *Neurocognitive effects of antipsychotic medications in patients with chronic schizophrenia in the CATIE Trial*. Arch Gen Psychiatry, 2007. **64**(6): p. 633-47.
30. Mishara, A.L. and T.E. Goldberg, *A meta-analysis and critical review of the effects of conventional neuroleptic treatment on cognition in schizophrenia: opening a closed book*. Biol Psychiatry, 2004. **55**(10): p. 1013-22.
31. Knowles, E.E., A.S. David, and A. Reichenberg, *Processing speed deficits in schizophrenia: reexamining the evidence*. Am J Psychiatry, 2010. **167**(7): p. 828-35.

32. Reilly, J.L., et al., *Antipsychotic drugs exacerbate impairment on a working memory task in first-episode schizophrenia*. Biol Psychiatry, 2007. **62**(7): p. 818-21.
33. Koreen, A.R., et al., *Depression in first-episode schizophrenia*. Am J Psychiatry, 1993. **150**(11): p. 1643-8.
34. Cosoff, S.J. and R.J. Hafner, *The prevalence of comorbid anxiety in schizophrenia, schizoaffective disorder and bipolar disorder*. Aust N Z J Psychiatry, 1998. **32**(1): p. 67-72.
35. Hafner, H., et al., *Depression, negative symptoms, social stagnation and social decline in the early course of schizophrenia*. Acta Psychiatr Scand, 1999. **100**(2): p. 105-18.
36. McGlashan, T.H. and W.T. Carpenter, Jr., *An investigation of the postpsychotic depressive syndrome*. Am J Psychiatry, 1976. **133**(1): p. 14-9.
37. Birchwood, M., et al., *Cognitive approach to depression and suicidal thinking in psychosis. 1. Ontogeny of post-psychotic depression*. Br J Psychiatry, 2000. **177**: p. 516-21.
38. Muller, J.E., et al., *Anxiety Disorders and Schizophrenia*. Current Psychiatry Reports, 2004. **6**(4): p. 255-261.
39. Michail, M. and M. Birchwood, *Social anxiety disorder in first-episode psychosis: incidence, phenomenology and relationship with paranoia*. Br J Psychiatry, 2009. **195**(3): p. 234-41.
40. Burt, D.B., M.J. Zembar, and G. Niederehe, *Depression and memory impairment: a meta-analysis of the association, its pattern, and specificity*. Psychol Bull, 1995. **117**(2): p. 285-305.
41. Snyder, H.R., *Major depressive disorder is associated with broad impairments on neuropsychological measures of executive function: a meta-analysis and review*. Psychol Bull, 2013. **139**(1): p. 81-132.
42. Castaneda, A.E., et al., *A review on cognitive impairments in depressive and anxiety disorders with a focus on young adults*. Journal of Affective Disorders, 2008. **106**(1-2): p. 1-27.
43. Potter, G.G. and D.C. Steffens, *Contribution of depression to cognitive impairment and dementia in older adults*. Neurologist, 2007. **13**(3): p. 105-17.

44. Wechsler, D., *Wechsler Adult Intelligence Scale - UK Third Edition (WAIS-IIIUK)*. 1997, Pearson: London.
45. Nelson, H.E. and J. Wilson, *National Adult Reading Test (NART)*. 1982, NFER-Nelson: Windsor, UK.
46. Wechsler, D., *Wechsler Test of Adult Reading (WTAR UK)*. 2001, Pearson: London.
47. Coughlin, A.K. and S.E. Hollows, *The Adult Memory and Information Processing Battery (AMIPB)*. 1985, St. James University Hospital: Leeds.
48. Warrington, E., *The Camden Memory Tests*. 1996, Routledge, Taylor & Francis Group: Abingdon, Oxford.
49. Reitan, R.M., *Trail Making Test*. 1992, Reitan Neuropsychology Laboratory: Mesa, AZ.
50. Burgess, P.W. and T. Shallice, *Hayling and Brixton Tests*. 1997, Pearson: London.
51. Golden, C., *Stroop Colour and Word Test*. 1978, Psychological Assessment Resources, Inc.: Lutz, FL.
52. Spielberger, C.D., et al., *Manual for the State-Trait Anxiety Inventory*. 1983, Palo Alto, CA: Consulting Psychologists Press.
53. Wechsler, D., *Test of Premorbid Functioning - UK Version (TOPFUK)*. 2011, Pearson Assessment: London.
54. Wechsler, D., *Wechsler Memory Scale - UK 4th Edition*. 2009, Pearson: San Antonio, TX.
55. Wechsler, D., *Wechsler Abbreviated Intelligence Scale - Second Edition (WASI-II)*. 2011, Pearson: London.
56. Grant, D.A. and E.A. Berg, *Wisconsin Card Sorting Test*. 1993, Psychological Assessment Resources, Inc.: Odessa, Florida.
57. Dickinson, D., M.E. Ramsey, and J.M. Gold, *Overlooking the obvious: a meta-analytic comparison of digit symbol coding tasks and other cognitive measures in schizophrenia*. Arch Gen Psychiatry, 2007. **64**(5): p. 532-42.

58. Hosenbocus, S. and R. Chahal, *A review of executive function deficits and pharmacological management in children and adolescents*. J Can Acad Child Adolesc Psychiatry, 2012. **21**(3): p. 223-9.
59. Clark, D.M. and A. Wells, *A cognitive model of social phobia*, in *Social Phobia: Diagnosis, Assessment and Treatment*. 1995, The Guildford Press: New York. p. 69-93.
60. Wells, C.E., *Pseudodementia*. Am J Psychiatry, 1979. **136**(7): p. 895-900.
61. Harvey, P.D., *Cognitive impairment in schizophrenia : characteristics, assessment, and treatment*. 2013, Cambridge: Cambridge University Press. xv, 324 p.
62. Knight, R.G., H.J. Waal-Manning, and G.F. Spears, *Some norms and reliability data for the State--Trait Anxiety Inventory and the Zung Self-Rating Depression scale*. Br J Clin Psychol, 1983. **22** (Pt 4): p. 245-9.
63. Kvaal, K., et al., *The Spielberger State-Trait Anxiety Inventory (STAI): the state scale in detecting mental disorders in geriatric patients*. Int J Geriatr Psychiatry, 2005. **20**(7): p. 629-34.
64. Ryan, J.J., et al., *Exploratory factor analysis of the Wechsler Abbreviated Scale of Intelligence (WASI) in adult standardization and clinical samples*. Appl Neuropsychol, 2003. **10**(4): p. 252-6.

APPENDIX 1: VERBATIM REPRODUCTION OF THE PATIENT'S PREVIOUS NEUROPSYCHOLOGICAL ASSESSMENT IN 2005

A note on the text:

The patient's neuropsychological assessment from 2005 is presented below verbatim, together with typos/incorrect spellings, which are indicated by '[sic]'. The patient's name, together with any other potentially identifiable information, has been anonymised.

REASON FOR REFERRAL:

Neuropsychological assessment of a 48-year-old right-handed gentleman, who has an approximately 10-year history of auditory hallucinations (currently in remission), persecutory delusions and deterioration of his personality. There is also a history of a road traffic accident in 2000 and [the patient] demonstrated executive and memory deficits when seen in the neuropsychiatry outpatient clinic in March 2005.

BACKGROUND:

[The patient] has an approximately 10-year psychiatric history. He apparently began hearing voices in the mid-1990s, but this remained untreated until his first psychiatric presentation in December 2000, when he was admitted to the [name of ward] at [name of hospital]. At that time, he was thought to be experiencing primarily a paranoid state, with secondary depressive features; he was treated with amisulpride. His mental state improved and he was discharged on 5th January 2001. [The patient] indicated during the current assessment that he no longer hears voices but had experienced a previous relapse when he stopped taking his medication.

In 2000, [the patient] was also involved in a road traffic accident. He was working at the time as a cycle messenger in London and was hit by a van whilst working. [The patient] reports having lost consciousness for about 30 seconds but remembers lying in the road and being helped up after the accident. He said that he had sustained a cut on the back of his head and was taken to hospital by ambulance, where the wound was stitched and he was discharged. He said that he required a couple of weeks off work that [sic] did not note any changes in his memory or concentration following the accident. In addition, he said that he had not experienced any anxiety about resuming riding his bike. He no longer works but had given up working as a result of his

psychiatric state, rather than due to any changes that he [sic] might have occurred following the accident. He described the voices that he had been hearing as developing slowly over time and after the accident they continued getting worse.

[The patient] does report a deterioration in his memory but feels that this has become poor since his hospital admission in late 2000, rather than occurring after the accident. He says that he will remember some events but generally not what has just happened. If he reads a paper or book then after about five minutes he will need to keep going back to re-read the same passages. He said that he is 'terrible' at remembering telephone messages and cannot remember the story line of television serials from one episode to another. He said that he gets confused when undertaking additions or subtractions and that this has also developed since his psychiatric admission. His partner, Robert Brent, helps him out with his finances although he will occasionally go to the shops on his own. [The patient] said that he no longer rides his cycle, having 'lost his nerve' since his psychiatric hospital admission.

[The patient]'s partner, [partner's name], commented that [the patient] now needs considerable encouragement to undertake everyday activities. [The patient's partner] has known [the patient] for 30 years. Currently, [the patient's partner] said that [the patient] undertakes less in terms of self-care activities such as keeping his fingernails clean or showering, but [the patient's partner] said that [the patient] does have good and bad days. [The patient's partner] said however that when [the patient's partner] is not around, he is not sure how well [the patient] copes with everyday activities, although he is reliable about taking his medication. [The patient's partner] said that [the patient] is very nervous about relating to people and this extends to for example, going to the post office or travelling by bus. When he is anxious he will stand making rocking movements or pace around. However, [the patient's partner] felt that [the patient] had improved over the last couple of years.

By way of education, [the patient] said that he had left school at the age of 16 and reports having taken three CSEs (in English Language, History and Religious Education). He then worked in the music business as a messenger for 15 years and then became a cycle messenger for [name of company], and worked for this group for many years.

[The patient's] current medication is risperidone 4mg daily, citalopram 40mg daily and procyclidine 5mg bd. In addition, [the patient] has non-insulin dependent diabetes for

which he takes metformin tablets. [The patient] said that his diabetes was first diagnosed in either 1999 or 2000. He says that he has greater difficulty controlling his diabetes when he is depressed. This is because he cannot be bothered to eat the right food or monitor his blood sugar levels. He says that he sees a specialist and a dietician at [name of hospital] for this.

BEHAVIOUR DURING ASSESSMENT:

[The patient] was seen on two separate assessment sessions. During both of these he appeared very anxious about the testing process and demonstrated at times quite marked akathisia, in terms of leg movements, which did appear to worsen then [sic] he was finding tasks particularly difficult. He would also sit and rock backwards and forwards when finding tasks difficult. However, he appeared adequately motivated to complete testing and was prepared to return for the second assessment session.

ASSESSMENT RESULTS:

(a) General Intellectual Ability:

[The patient] was assessed on 11 subtests of the WAIS-III. On the basis of these he obtained a prorated verbal IQ of 87 (low average range) and a prorated performance IQ of 75 (borderline range). Although the difference between his verbal and performance IQ was suggestive of a statistically significant difference, a discrepancy of this size is not thought to be clinically abnormal.

[The patient] obtained a verbal comprehension index of 93 (average range) and a perceptual organisation index of 82 (low average range). Again although the difference between these two scores was statistically significantly different, it is not of a clinically abnormal magnitude. [The patient's] working memory index was 75 (borderline range) and his processing speed index was 68 (extremely low range). Whilst both [the patient's] perceptual organisation index and his verbal comprehension index were significantly higher than his processing speed index from a statistical point of view, it was the discrepancy between his verbal comprehension index and processing speed index that tended to suggest a clinically significant relative weakness in his processing speed index.

In terms of obtaining estimates of [the patient]'s premorbid level of functioning, he was assessed on both the National Adult Reading Test and the Wechsler Test of Adult

Reading. Both of these measures yielded premorbid estimates of general intellectual ability in the average range. When comparing WTAR-predicted WAIS-III scores with his current verbal and performance IQs, although both of his current IQs were significantly lower than the predicted levels, it was the discrepancy between his predicted and current performance IQ that appeared to represent a clinically significant current level of underfunctioning. It is likely that this discrepancy between predicted and current performance IQ scores reflects his particularly slow processing speed. This is to some extent substantiated by the observation that his performance on the untimed matrix reasoning subtest was in the average range (scaled score 9; IQ equivalent 95).

(b) Memory:

[The patient] was assessed on the four memory subtests from the Adult Memory and Information Processing Battery (AMIPB) and also the Short Recognition Memory Tests for Words and Faces from the Camden Test Battery.

[The patient]'s performance on tests of memory functioning were [sic], to some extent, rather patchy. In terms of his ability to recall a short story, his immediate recall was below average (10-25th %ile) but his delayed recall was well below average (10th %ile). His percentage retained score fell below the 10th %ile. In terms of his ability to learn a list of words, presented over five trials, he achieved a total learning score that fell in the below average range (10-25th %ile) and he showed a rapid plateau in terms of the number of words retained across successive trials. His delayed recall of the initial list was impaired and his one trial learning of the distraction list was at the lower end of the average range. His forced choice recognition of words was impaired (below the 5th %ile) but he had initially misunderstood some of the task instructions.

[The patient] produced a broadly accurate copy of the AMIPB complex figure but produced this copy large [sic] without a systemic [sic] plan. His immediate recall was at the 50th %ile but he showed marked forgetting of the figure over the 30 minute delay such that his absolute level of delayed recall was well below average (below the 10th %ile) but his percentage retention score was impaired (below the 2nd %ile). His ability to learn an abstract design, presented over five trials was below average (10-25th %ile) although he showed a perfect retention of the figure after an intervening distraction trial. However, his one trial learning of the distraction trial itself was impaired (below

the 2nd %ile). However, his forced choice recognition of faces was at ceiling level and he made no errors on the test.

(c) Language:

[The patient]'s reading ability, as noted earlier, is consistent with an average level of general intellectual ability. He obtained a score of 21/30 on the Graded Naming Test, which is in line with his current reading ability and does not suggest any acquired dysnomic difficulties. In addition, his verbal fluency, as assessed on the FAS test, yielded a number of words that was consistent with his reading ability and did not therefore suggest any difficulties in word generation.

(d) Executive Functions:

As noted above, [the patient]'s performance on the verbal fluency test was in line with the level predicted on the basis of his current reading ability and did not suggest any acquired impairments of verbal fluency. On the WAIS-III similarities subtest, [the patient] obtained an age scaled score that had an IQ equivalent in the low average range (i.e. 80). This suggests some degree of relative weakness in terms of abstract thinking compared to his ability to retrieve semantic information as assessed by the Information subtest where his IQ equivalent was 105. Although [the patient] made an error on Part B of the trail making test, his completion time on Part B was not disproportionately slower to that seen on Part A, and whilst suggesting very mild difficulties in self-monitoring, did not suggest any particular difficulty with divided attention that could be attributed to a dysexecutive syndrome.

However, [the patient] did have more pronounced difficulties on the Stroop colour-word interference test, where his completion time and score placed him below the 2nd %ile. In addition on the Hayling sentence completion test, while his completion times for both the sensible and unconnected sentences were classified as moderate average, he made a number of both connected and somewhat connected error responses, his score on the error measure being classified as Poor. Thus he appeared to have some difficulty in response inhibition on this measure. On the Brixton spatial anticipation test, he made a total of 29 errors with his score overall being classified as abnormal.

Thus, there is mild evidence for difficulties with abstract reasoning but more marked difficulties on tests of response inhibition, sequence detection and rule following.

CONCLUSIONS:

In terms of general intellectual ability, [the patient] is currently functioning in the low average to borderline range, although a purer measure of his verbal intellectual skills, which does not rely on working memory measures, places him more clearly in the average range. His current IQ scores represent a significant degree of underfunctioning compared to his premorbid, reading-based estimates, although the discrepancy is more marked for his performance than his verbal IQ, and this is probably attributable to his slowed processing speed as recorded during the current assessment. [The patient]'s memory functioning would appear to be somewhat inconsistent across tests, and on a number of measures he performed in the below average to well below average range. No difficulties were detected in terms of naming or verbal fluency, but assessment of executive functions revealed mild relative difficulty on a test of abstract reasoning and more pronounced deficits on measures of response inhibition, rule detection and sequence following.

[The patient] is currently awaiting an MRI scan. It will of course be important to interpret the results of the current assessment in light of any findings from that scan, but it is of note that neither he nor his partner attribute any of his current cognitive or psychological difficulties to the apparently mild head injury which he sustained in 2000. It would therefore be difficult to attribute the current executive function difficulties, along with his slowed processing speed, to that accident and it may be more likely that his psychiatric history and its treatment account for his current cognitive profile. In addition it is possible that poor control of his diabetes may place him risk [sic] for poor cognitive functioning, especially in terms of memory. However, the current assessment will serve as a good baseline against which to evaluate any suspected future change in [the patient]'s cognitive profile.

1)	<u>WAIS-III</u>			
	Verbal	Age Scaled	Performance	Age Scaled
	Vocabulary	9	Picture Completion	5
	Similarities	6	Matrix Reasoning	9
	Arithmetic	6	Block Design	7
	Digit Span	7	Digit Symbol Coding	3
	Information	11	(Symbol Search)	(4)
	(Letter Number Sequencing)	(5)		
2)	<u>Pro-Rated</u>	VIQ = 87	Pro-Rated	PIQ = 75
		VCI = 93		POI = 82
		WMI = 75		PSI = 68
3)	<u>NART-R</u>			
	Error Score	= 18		
	Predicts WAIS-R	FSIQ = 108		
		VIQ = 107		
		PIQ = 108		
4)	<u>AMIPB</u>			
	a) <u>Story Recall</u>	<u>Raw Score</u>	<u>Percentile</u>	
	Immediate	25	10-25	
	Delayed	17	10	
	% Retained	68	<10	
	b) <u>List Learning</u>			
	Total A1-A5	39	10-25	
	A6	5	2 nd	
	B	4	25	
	Intrusions	0	75+	
	c) <u>Figure Recall</u>			
	Copy	97.5%	25-50	
	Immediate	76.92%	50	
	Delayed	38.46%	<10	

	% Retained	50%	<2 nd	
	d) <u>Design Learning</u>			
	Total A1-A5	25	10-25	
	A6	9	90	
	B	0	<2 nd	
	Intrusions	3	90	
5)	<u>Camden Short Recognition Memory Test</u>			
	Words	Raw Score	= 20/25	
		%ile	<5 th %ile	
	Faces	Raw Score	= 25/25	
		%ile	90+	
6)	<u>Trail Making Test</u>			
	Part A	52 sec.	10-25 th %ile	
	Part B	132 sec.	10-25 th %ile	1 error
7)	<u>Stroop Test</u>			
	Colour Words	Score =	112/112	
		Time =	69 sec.	
	Colour Words Interference	Score =	45/112	
		%ile =	< 2 nd	
		Time =	260 sec.	
8)	<u>Graded Naming Test</u>			
	Score =	21/30	50-75 th %ile	
9)	<u>Word Fluency</u>			
	F	19		
	A	9		
	S	11		
	Total	39		
	Predicted by NART	= 44		
10)	<u>Hayling Sentence Completion Test</u>			
	Sensible Completion			

	Time Scaled Score =	5 = "Moderate Average"	
	Unconnected Sentences		
	Time Scaled Score =	5 = "Moderate Average"	
	Hayling 2 errors Scaled Score =	3 = "Poor"	
	Overall Scale Score =	4 "Low Average"	
<u>11)</u>	<u>W.T.A.R.</u>		
	Raw Score	= 38	
	Standard Score	= 99	
	Predicted WAIS-III	VIQ	100 (95% c.i. 82-120)
		PIQ	102 (95% c.i. 77-127)
<u>12)</u>	<u>Brixton Spatial Anticipation Test</u>		
	Errors = 29		
	Scaled Score = 2 = "Abnormal")		

APPENDIX 2: FULL DESCRIPTION OF THE MEASURES USED IN THE PRESENT ASSESSMENT

THE SPIELBERGER STATE AND TRAIT ANXIETY INVENTORY [52]

The STAI was administered due to the patient's well-documented history of chronic anxiety – and in particular, his chronic history of social anxiety; his level of anxiety at the time of testing was therefore of interest. The STAI comprises two self-report questionnaires. One measures state anxiety (s-anxiety; form Y-1) – that is, the anxiety experienced at the time the scale is completed, whilst the trait anxiety (t-anxiety; form Y-2) questionnaire indexes one's general tendency towards anxiety. Each scale consists of 20 items, with responses scored from 1 ('almost never') to 4 ('almost always') via a Likert scale; thus, the total score for each can range from 20 to 80, with higher scores indicating greater anxiety. A cut-off point of 39-40 has been suggested for inferring clinical significance in each scale [STAI; 52], though others have suggested a higher cut-off of 54-55 [62]. Internal reliability coefficients for the scale range from 0.86 to 0.95, whilst test-retest correlations, across a two-month interval, range from 0.65 to 0.75 [63].

THE TEST OF PREMORBID FUNCTIONING – UK EDITION [TOPFUK; 53]

The TOPF^{UK} consists of a list of 70 words that have atypical grapheme to phoneme translations. The examinee must read each word from the list aloud in turn, with the words becoming increasingly phonetically irregular as one progresses down the list. As implied by its name, this measure is used to estimate premorbid intellectual and memory abilities, as the ability to identify and articulate words is thought to be relatively resistant to the effects of brain-injury and/or dementia. The test's scoring manual provides age-corrected standard scores with a mean of 100 and standard deviation (SD) of 15; this standard score was used here to compare the examinee's predicted and observed memory abilities.

THE WECHSLER MEMORY SCALE – UK 4TH EDITION [WMS-IVUK; 54]

The WMS-IV^{UK} was used to comprehensively assess the patient's memory functioning. It is a widely-used memory test battery and comprises 10 subtests and assesses a range of memory processes, generating five domain-related indices in line with theories fractionating memory function: the auditory memory index (AMI), the visual memory

index (VMI), the visual working memory index (VWMI), the immediate memory index (IMI) and the delayed memory index (DMI). Tables A1-A5 present descriptions of the WMS-IV^{UK} indices and their incorporated subtests.

Table A1: Descriptions of the auditory memory index and its incorporated subtests

Index/Subtest	Description
Auditory Memory Index	This index indicates the examinee's ability to remember orally-presented information.
Logical Memory I	This subtest assesses narrative memory under a free-recall condition. Two short stories are orally presented. The examinee is asked to retell each story from memory immediately after hearing it.
Logical Memory II	The delayed condition assesses long-term narrative memory with free-recall and recognition tasks. The examinee is asked to retell both stories from the immediate condition. Then, the examinee is asked yes/no questions about both stories.
Verbal Paired Associates I	This subtest assesses verbal memory for associated word-pairs. After 14 word-pairs are read to the examinee, the first word of each pair is read, and the examinee is asked to provide the corresponding word. There are four trials of the same list in different orders.
Verbal Paired Associates II	The delayed condition assesses long-term recall for verbally-paired information with cued-recall and recognition tasks. The examinee is orally presented with the first word of each pair learned in the immediate condition and asked to provide the corresponding word. The examinee is then read a list of word-pairs and asked to identify each as either one of the word-pairs he or she was asked to remember, or a new word-pair.

Table A2: Descriptions of the Visual Memory Index and its incorporated subtests

Index/Subtest	Description
Visual Memory Index	This index indicates the examinee's ability to remember visually-presented information.
Designs I	This subtest assesses spatial memory for unfamiliar visual material. The examinee is shown a grid with 4-8 designs on a page for 10 seconds, which is then removed from view. The examinee then selects the designs from a set of cards and places the cards in a grid in the same place as previously shown.
Designs II	The delayed condition assesses long-term spatial and visual memory with free-recall and recognition tasks. First, the examinee is asked to recreate the pages shown in the immediate condition with the cards and grid. Then, he or she is shown a series of grids and asked to select the two designs that are correct and in the same place as on the pages shown in the immediate condition.
Visual Reproduction I	This subtest assesses memory for non-verbal visual stimuli. A series of five designs is shown, one at a time, for 10 seconds each. After each design is presented, the examinee is asked to draw the design from memory.
Visual Reproduction II	The delayed condition assesses long-term visual-spatial memory with free-recall and recognition tasks. First, the examinee is asked to draw the designs shown during the immediate condition, from memory in any order. Second, the examinee is asked to choose which of six designs on a page matches the original design shown during the immediate condition.

Table A3: Descriptions of the Visual Working Memory Index and its incorporated subtests

Index/Subtest	Description
Visual Working Memory Index	This index indicates the examinee's capacity to remember and manipulate visually-presented information in short-term memory storage.
Spatial Addition	This subtest assesses visual-spatial working memory, using a visual addition task. The examinee is shown, sequentially, two grids with blue and red circles. The examinee is then asked to add or subtract the location of the circles, based on a set of rules.
Symbol Span	This subtest assesses visual working memory using novel visual stimuli. The examinee is briefly shown a series of abstract symbols on a page and then asked to select the symbols from an array of symbols, in the same order they were presented on the previous page.

Table A4: Descriptions of the Immediate Memory Index and its incorporated subtests

Index/Subtest	Description
Immediate Memory Index	This index indicates the examinee's ability to remember both visually- and orally-presented information immediately after it is presented.
Logical Memory I	This subtest assesses narrative memory under a free-recall condition. Two short stories are orally presented. The examinee is asked to retell each story from memory immediately after hearing it.
Verbal Paired Associates I	This subtest assesses verbal memory for associated word-pairs. After 14 word-pairs are read to the examinee, the first word of each pair is read, and the examinee is asked to provide the corresponding word. There are four trials of the same list in different orders.
Designs I	This subtest assesses spatial memory for unfamiliar visual material. The examinee is shown a grid with 4-8 designs on a page for 10 seconds, which is then removed from view. The examinee then selects the designs from a set of cards and places the cards in a grid in the same place as previously shown.
Visual Reproduction I	This subtest assesses memory for non-verbal visual stimuli. A series of five designs is shown, one at a time, for 10 seconds each. After each design is presented, the examinee is asked to draw the design from memory.

Table A5: Descriptions of the Delayed Memory Index and its incorporated subtests

Index/Subtest	Description
Delayed Memory Index	This index indicates the examinee's ability to remember both visually- and orally-presented information after a 20-30 minute delay.
Logical Memory II	The delayed condition assesses long-term narrative memory with free-recall and recognition tasks. The examinee is asked to retell both stories from the immediate condition. Then, the examinee is asked yes/no questions about both stories.
Verbal Paired Associates II	The delayed condition assesses long-term recall for verbally-paired information with cued-recall and recognition tasks. The examinee is orally presented with the first word of each pair learned in the immediate condition and asked to provide the corresponding word. The examinee is then read a list of word-pairs and asked to identify each as either one of the word-pairs he or she was asked to remember, or a new word-pair.
Designs II	The delayed condition assesses long-term spatial and visual memory with free-recall and recognition tasks. First, the examinee is asked to recreate the pages shown in the immediate condition with the cards and grid. Then, he or she is shown a series of grids and asked to select the two designs that are correct and in the same place as on the pages shown in the immediate condition.
Visual Reproduction II	The delayed condition assesses long-term visual-spatial memory with free-recall and recognition tasks. First, the examinee is asked to draw the designs shown during the immediate condition, from memory in any order. Second, the examinee is asked to choose which

of six designs on a page matches the original design shown during the immediate condition.

THE 2-SUBTEST VERSION OF THE WECHSLER ABBREVIATED SCALE OF INTELLIGENCE –2ND EDITION [WASI-IIUK; 55]

The WASI-II^{UK} was used to give an indication of the examinee’s general cognitive abilities. This measure offers the option of a 4- or a 2-subtest version; although the former is considered to provide a more reliable indication of current cognitive ability [52], it takes 30 minutes to complete and, as time was at a premium, the 2-subtest version was used here. The 2-subtest version yields a single score, that of Full-Scale IQ (FSIQ), based on performance within the Vocabulary and Matrix Reasoning subtests. Table A6 presents descriptions of the vocabulary and matrix reasoning subtests of the WASI-II^{UK}.

Table A6: Descriptions of the Vocabulary and Matrix Reasoning subtests of the WASI-II^{UK}

Subtest	Description
Vocabulary	The Vocabulary subtest has 31 items, including 3 picture items and 28 verbal items. For picture items, the examinee names the object presented visually. For verbal items, the examinee defines words that are presented visually and orally. Vocabulary is designed to measure an examinee's word knowledge and verbal concept formulation. It also measures an examinee's crystallised intelligence, fund of knowledge, learning ability, long-term memory and degree of language development. Other abilities that may be used by the examinee during this task include auditory comprehension and verbal expression.
Matrix Reasoning	The Matrix Reasoning subtest has 30 items. The examinee views a series of incomplete matrices and completes each one by selecting the correct response option. The subtest taps fluid intelligence, broad visual intelligence, classification and spatial ability, knowledge of part-whole relationships, simultaneous processing and perceptual organisation.

The WCST is one of the most commonly-used instruments for the assessment of executive function and is considered a significant measure of cognitive flexibility, attention and impulsivity. The test assesses abstract reasoning, the subject's ability to generate problem-solving strategies in response to changing conditions and may be regarded, therefore, as a measure of flexibility of thought. Briefly, the WCST test consists of 128 paper cards containing geometric designs that vary in colour, form and number. The subject is given four cards and then asked to sort the remaining deck of cards by colour, form or number, but is not instructed as to how to do so. Thus, the subject is required to infer the correct sorting principles from limited feedback from the examiner, who only tells the participant, following each individual card-sort, whether the sorting is correct or incorrect.

APPENDIX 3: SCHEMATIC OVERVIEW OF MEASURES ADMINISTERED AND THEIR SEQUENCING/TIMINGS

Table A7 presents a schematic overview of the measures administered as part of the assessment, together with the sequence in which they were administered and the time taken to complete each.

Table A7: Schematic overview of measures administered and their sequencing/timings

Session	Order of tests (and timings)
1	<p>Informal interview (15 minutes)</p> <p>State and Trait Anxiety Inventory (STAI) (15 minutes)</p> <p>Test of Premorbid Functioning (TOPF^{UK}) (3 minutes)</p> <p>Wechsler Memory Scale – UK 4th Edition (WMS-IV^{UK}) (Subtests 1-5) (40 minutes)</p>
2	<p>WMS-IV^{UK} (subtests 6-10) (35 minutes)</p> <p>Wechsler Abbreviated Intelligence Scale – 2nd Edition (WASI-II^{UK}) (30 minutes)</p> <p>Wisconsin Card-Sorting Test (WCST) (45 minutes)</p>

THE STAI

The patient's state (s-anxiety) was 67 and his trait anxiety (t-anxiety) was 75, indicating considerable anxiety. Of most interest for the present purposes was his s-anxiety – that is, the degree to which he was experiencing anxiety *in the moment*, during the testing sessions. His score of 67 breached both the more liberal (39-40) and the more conservative (54-55) cut-offs for inferring the presence of clinically-significant symptoms. Furthermore, his s-anxiety score was substantially higher than for adult males of the same age within the general population ($M = 34.51$; $SD = 10.34$), placing him within the top 1% [64]. Even relative to other male neuropsychiatric patients (i.e. those with major depression, an anxiety disorder, schizophrenia, brain damage), his scores were high, with mean s-anxiety across other neuropsychiatric groups equaling 47.74 ($SD = 13.24$); the patient's s-anxiety score therefore places him within the top 10% of neuropsychiatric patients. His (very) high s-anxiety is consistent with his apparent apprehension and anxiety around his presenting for and attendance at the sessions. This is of considerable importance, given the potentially deleterious impact of high levels of anxiety on one's ability to concentrate and allocate attentional resources to ongoing tasks, such as those completed in the current assessment. His trait anxiety score was even greater than his state anxiety score, consistent with the chronic anxiety documented in his care record. It is therefore likely that the patient's chronically elevated anxiety also interferes with his cognitive and memory functioning in everyday life, impairing his ability to follow conversations and remember why he has entered the kitchen, for example.

THE WMS-IV^{UK}

The patient's performance on the WMS-IV^{UK} indicated substantial and global memory impairment. The examinee's performance across all indices of the WMS-IV^{UK} fell into the extremely low range. Table A8 presents the results of the WMS-IV^{UK}.

Table A8: Results of the WMS-IV^{UK}

Index/Subtest	Index / Scaled Score	95% Confidence Interval	Percentile Rank ¹	Descriptive Category
Auditory Memory Index	67	62-75	1	Extremely Low
Logical Memory I	2			
Logical Memory II	3			
Verbal Paired Associates I	5			
Verbal Paired Associates II	8			
Visual Memory Index	57	53-64	0.2	Extremely Low
Designs I	8			
Designs II	2			
Visual Reproduction I	2			
Visual Reproduction II	1			
Visual Working Memory Index	70	65-79	2	Borderline
Spatial Addition	5			
Symbol Span	5			
Immediate Memory Index	61	57-69	0.5	Extremely Low
Logical Memory I	2			
Verbal Paired Associates I	5			
Designs I	8			
Visual Reproduction I	2			
Delayed Memory Index	56	52-65	0.2	Extremely Low
Logical Memory II	3			
Verbal Paired Associates II	8			
Designs II	2			
Visual Reproduction II	1			

The AMI: This measures the ability to remember orally-presented information. The examinee's AMI score was extremely low, compared to others his age. There was significant inconsistency in performance on specific measures within this domain, warranting a closer look. Thus, his performance on the verbal paired associates II subtest was a statistically-significant strength, indicating that he has an improved ability to remember word-pairs 20-30 minutes after presentation. On the other hand, a relative weakness was the score he obtained on logical memory I – that is, the ability to recall verbal information that is conceptually-organised and semantically-related immediately after hearing it.

The VMI: The VMI measures the ability to process, analyse and recall visually-presented information. The examinee's score placed him within the extremely low ability range. However, there was significant inconsistency in his performance on specific measures within this domain, warranting a closer look at his scores. The examinee's performance in designs I was significantly better than his average performance on the other subtests within this index; the scale of the discrepancy between his improved score on the former relative to his mean VMI performance is shared by only 1% of the population. Thus, a relative strength of his is his immediate recall of unfamiliar visual material. In contrast, the examinee's performance on designs II was worse, suggesting that his long-term recall is poor. Interestingly, his ability to recognise unfamiliar visual material from long-term memory (designs II recognition) was significantly better and was equivalent to the performance of 26-50% of the general population. These data are consistent with the examinee's being able to encode information into long-term memory but experiencing difficulty with its subsequent retrieval; indeed, when asked during the testing sessions whether he had any methods via which he was trying to remember material, the examinee responded in the negative.

The VWMI: The VWMI evaluates the temporary storage and manipulation of visual information within a virtual mental notepad. The examinee performed in the borderline ability range; his performance here is shared by only 2% of the general population.

The IMI: The IMI is a measure of the ability to recall verbal and visual information immediately after the stimuli have been presented. Compared to other individuals his age, the examinee performed in the extremely low range, his performance being shared

with just 0.5% of the general population. Within this index, the examinee exhibited a strength on the designs I subtest; thus, his ability to retrieve unfamiliar visual information from short-term memory was superior to his performance on the other IMI subtests.

The DMI: The DMI indexes the ability to recall verbal and visual information after a 20- to 30-minute delay. As per his performance on the other indices, the examinee was again placed within the extremely low ability category, his score being shared with only 0.2% of the general population. There was inconsistency amongst the examinee's subtest scores within this index, which merits further discussion. His performance on the verbal paired associates II subtest was a strength, meaning that, relatively speaking, he was more able in terms of the long-term recall of pairs of words.

THE TOPF^{UK}

The patient's TOPF^{UK} raw score was 56, placing his premorbid estimates in the average to high average range. His estimated premorbid scores for WMS-IV^{UK} indices were uniformly substantially greater than his obtained scores. The discrepancies between his estimated and observed WMS-IV^{UK} scores were so great, in fact, as to be statistically implausible – indeed, fewer than 0.09% of the general population exhibit such discrepancies between their expected and observed scores. This calls into question the validity of his WMS-IV^{UK} performance. Table A9 presents the results of the TOPF^{UK}.

Table A9: Discrepancies between estimated premorbid and current obtained scores on the WMS-IV^{UK}, based on TOPF^{UK} raw score of 56

Index	Obtained Score	Estimated Premorbid Score	Discrepancy (Obtained minus Estimated)	Percentage of Normative Population Expected to Exhibit a Lower Discrepancy Score: Point Estimate
Immediate Memory Index	61	107	-46	< 0.06
Delayed Memory Index	56	107	-51	< 0.09
Visual Working Memory Index	67	110	-43	< 0.06

THE WASI-II^{UK}

The examinee's performance on the WASI-II^{UK} placed him within the borderline range for FSIQ. According to his performance, there is a 95% chance that his 'true' IQ score lies somewhere between 71 and 84. Considering that one criterion for intellectual disability is an IQ below 70, this may suggest that he is on the borderline for such a diagnosis. However, even the most cursory of meetings with the patient reveals that he is clearly not intellectually challenged, and neither has the possibility ever been queried; indeed, his very low IQ score represents a further challenge to the validity of his overall performance in the present assessment. Table A10 presents the results of the WASI-II^{UK}.

Table A10: Results of the WASI-II^{UK}

Scale/Subtest	T Score	Composite Score	Percentile Rank	Confidence Interval	Descriptive Category
Full Scale-2	72	76	5	71-84	Borderline
Matrix Reasoning	39				
Vocabulary	33				

THE WCST

The patient's performance on the WCST demonstrated that his executive functioning was significantly impaired. The large number of errors he made meant his performance was shared with the lowest 5% of the general population. Similarly, the very high numbers of perseverative responses and errors he made were shared with less than 1% of the population. He demonstrated significant difficulty in appreciating the need to learn using corrective feedback; indeed, his low Learning to Learn score was shared with less than 1% of the (worst) performers on this task in the general population. Overall, the patient's WCST performance was similar to that expected for individuals with frontal lobe lesions. Table A11 presents the results of the WCST.

Table A11: Results of the WCST

	Raw Score	Standard Score	T Score	Percentile Score/Range
Number of Trials Administered	128			
Total Number Correct	55			
Total Number of Errors	73	71	31	3
Percent Errors	57	72	31	3
Perseverative Responses	82	59	23	< 1
Percent Perseverative Responses	64	56	21	< 1
Perseverative Errors	65	60	23	< 1
Percent Perseverative Errors	51	57	21	< 1
Non-Perseverative Responses	8	108	55	70
Percent Non- Perseverative Responses	6	115	60	84
Conceptual Level Responses	37			
Percent Conceptual Level Responses	29	74	33	4
Number of Categories Completed	2			6-10
Trials to Complete First Category	19			6-10
Failure to Maintain Set	1			> 16
Learning to Learn	-28			≤ 1

CLINICAL CASE STUDY 2

'I'M FEELING REALLY QUITE FINE'

COGNITIVE-BEHAVIOURAL THERAPY VIA GRADED
EXPOSURE FOR ANXIETY/AVOIDANCE IN THE CONTEXT
OF PSYCHOSIS IN A 33-YEAR-OLD MAN

SUPERVISED BY DR. ABIGAIL CLARK

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I. LITERATURE REVIEW

I.I) PSYCHOSIS/SCHIZOPHRENIA

Schizophrenia is a chronic and severe mental disorder that affects an individual's thoughts, emotions and behaviour [1]. The symptoms typically emerge between the ages of 16 and 30 and fall into three main domains: (i) positive symptoms; (ii) negative symptoms and; (iii) cognitive symptoms. 'Positive symptoms' refer to thoughts and behaviours not typically observed in healthy people, such as hallucinations, delusions and disordered thinking. 'Negative symptoms' refer to disruptions of normal thoughts and behaviours, including 'flat affect' (e.g., reduced expression of emotions via facial expression or voice tone), attenuated feelings of pleasure, social withdrawal and reduced speech. Cognitive symptoms refer to a range of neuropsychological deficits often observed in schizophrenia, such as poor attention and memory, slow processing speed and impairments in executive functions (e.g., planning, inhibitory control, cognitive flexibility) [2].

It has been estimated that over half a million people in the UK, and around 25 million people worldwide, exhibit symptoms of psychosis [3, 4]. The potential for recovery is often reduced by the high levels of distress that can result from persisting psychotic symptoms, stigma and social exclusion [5, 6]. Indeed, surveys suggest that up to 60 percent of those with schizophrenia may be moderately to severely socially disabled [e.g., 7] – the term 'social disability' including an inability to function at work, in relationships and in carrying-out normal activities of daily living. It has been estimated that 80 percent are unemployed [8]. The lifetime risk of committing suicide is five percent, with up to 13 percent showing moderate to severe suicidal behaviour [9].

I.II) ANXIETY IN PSYCHOSIS/SCHIZOPHRENIA

Those with psychosis have an increased prevalence of anxiety, across all stages of the illness, relative to the general population [e.g., see reviews by 10, 11]. Indeed, anxiety is closely related to psychotic symptoms and may play a causal role in the onset and maintenance of psychosis, as well as arising in response to an episode and can be thus thought of as a central aspect of the disorder [12-14]. Although the reasons for severe social disability in the disorder appear complex and multifactorial [15], anxiety is at least partly attributable: thus, anxious avoidance can reduce the person's efficacy with

regards to entering and remaining in social situations, having clear implications for their ability to find and maintain work, for example [16].

I.III) ADDRESSING THE SYMPTOMS OF PSYCHOSIS/SCHIZOPHRENIA

The positive symptoms of psychosis have traditionally been thought of as untreatable via psychological approaches, although this position has changed markedly in recent decades [17]. Delusions and hallucinations have increasingly come to be viewed as occurring on a continuum with normality [18] and, consequently, as potentially susceptible to change via adaptations to cognitive-behavioural therapy (CBT) for anxiety and depression.

A number of modifications are recommended when applying CBT to psychosis (CBTp), with engagement and the establishment of a therapeutic alliance considered particularly important [16]. To achieve this, Kuipers *et al.* [17] emphasise keeping sessions non-aversive, which may include having shorter sessions than may be typical with other patient groups, as well as being more flexible / not pursuing session agendas too rigidly. It is generally held that therapists take up to six sessions to conduct a thorough assessment and develop a formulation collaboratively with the patient. This then allows for the development of a person-centred intervention, taking into account any cognitive deficits.

Given the importance of anxiety in the development and maintenance of psychotic symptoms [19], its reduction (and associated distress) is often a primary goal in CBT for psychosis, with such interventions often found to be effective [e.g., 16, 20-22]. With phobic symptoms, this would typically involve the construction of a hierarchy of feared scenarios, followed by a graded exposure programme working up each aspect of this hierarchy – via the modifications described above.

Randomised controlled trials (RCTs) have supported the application of CBTp, much of this evidence coming from four research groups in the United Kingdom: (i) Garety, Fowler and Kuipers in London and East Anglia; (ii) Tarrier and Bentall in Manchester; (iii) Kingdon and Turkington in Southampton and Newcastle and; (iv) Birchwood and colleagues in Birmingham [19]. These RCTs, reporting beneficial effects of CBT for psychosis, have been included in a series of meta-analyses [e.g., 23, 24-26]. Although these studies have varied in their approach and findings, there is an overall effect-size

of around 0.37 (i.e. between the small-moderate ranges), the largest effects found for persistent positive symptoms¹.

Evidence such as the above has led to CBT being used as standard practice to address the symptoms of psychosis. Thus, the most recent National Institute for Health and Care Excellence (NICE) guidelines for psychosis [CG178; 27] recommends cognitive-behavioural therapy (CBT) as a first-line intervention.

II. THE PATIENT

The patient, 'Adesola' (not his real name), was a 33-year-old Black-British male (of West African descent) who was a patient at a Community Mental Health Team (CMHT) for Psychosis. He had a diagnosis of schizophrenia, was unemployed and lived with a cousin Adesola spent a considerable amount of time working on a manuscript in which he outlined his thoughts and anxieties concerning the structure of the universe. He was on a regular dose of clozapine.

III. THE PRESENTING PROBLEM

Adesola was referred for psychological intervention due to a chronic anxiety about the safety of 'tall towers' (which he defined as those greater than 20 storeys), such as blocks of flats, the Shard, etc. His particular concern was that the foundations and general architecture of such structures were inadequate, such that they were vulnerable to potential collapse – and, more specifically, that, due to certain metaphysical 'powers' that he had, his proximity to tall buildings might act as a trigger for their collapsing. Adesola stated that, when near to such buildings, he hears voices telling him to 'go back, go back!' (i.e. to escape the situation), as the voices have a 'premonition that something bad will happen'. Adesola described catastrophic mental images he had of such towers collapsing, which seemed reminiscent of the attack on the twin towers in New York on the 11th September, 2001. Understandably, this made Adesola anxious about walking near tall buildings, resulting in avoidant behaviour: 'I have to be obedient in case something happens'. He was in fact so obedient that he rarely left his

¹ It should be noted that all the aforementioned trials included participants who had been prescribed anti-psychotic medication.

home, stating that the voices only allow him to go somewhere if/when there is no alternative (e.g., attending a medical/psychology appointment).

IV. BACKGROUND

As stated above, Adesola believed that he had been endowed with various metaphysical 'gifts', chief amongst these being an ability to hear the voices of 'everyone in the world'. As a result of this ability, Adesola referred to himself as 'the boy in the airwaves'. Adesola also stated that he was Jesus Christ and the President of the World, that he had 'mutant abilities' and that he could 'invent anything'. He said he was grateful for these powers and that they 'had given [him] a reason to live'.

Adesola had developed a relatively complex belief system around the voices, saying that there were 'good ones' and 'bad ones'. The former he labelled 'spirits', stating that 70 percent of which are older ladies and 30 percent being teenagers who had become his 'pals'. Some of Adesola's beliefs were rather grandiose. For example, he stated that the spirits were concerned with ridding the world of evil and that, as Jesus Christ, they were helping him in his destiny to save the world. Adesola said that he often speaks about moral and political issues with the spirits (e.g., 'whether it is acceptable to be mean to others'). Adesola sought advice on such issues as he was deeply focused on ensuring that, after death, he goes to Heaven. He felt compelled to obey them when they instructed him to do/not do certain things and said that if he does not obey them, they accuse him of being disrespectful and rude. Adesola felt that he had no control over the voices and described being dependent on them to tell him what to do. Regarding the 'bad' voices, Adesola described them as 'yobs' and being 'really horrible': 'all they talk about is evil things like murder... they always cause problems... they are bullies'.

Adesola believed the spirits to be a positive influence in his life, trying to both keep him, as well as other people, safe. One of their principal concerns was the potential collapse of tall towers, due to Adesola's superhuman powers; he thus believed that the voices were trying to protect anyone that may be in such buildings. He was also concerned that, due to his celebrity status, when out in public, he would be recognised by others as 'the boy in the airwaves', thinking that around 20 percent of people on the street could potentially recognise him. Of these people, he felt that whilst around half would be supportive, the other half would try to intimidate and/or attack him – for example, by stabbing him (he had not been attacked whilst out in public). Notably, he

said that his confidence ‘vanishes’ around strangers, leading him to be fearful in their presence. Overall, Adesola seemed to have a strong sense of the world and other people as being dangerous. He acknowledged his life as being restricted, but felt that this was preferable to either becoming harmed himself or his presence causing harm to others. He thus had a high sense of responsibility and morality.

Adesola had challenging relationships with members of his family, in particular with his mother (with whom he occasionally lived, during times when she returned from West Africa). Specifically, Adesola reported that he experiences ‘mental abuse’ from his family and described his mother as ‘constantly shouting and complaining’. He said that his mother ‘put him under pressure’ to get a job, that his family in general did not agree that he had a mental health problem and that overall, he found interactions with them to be stressful. When asked why he had not chosen to find somewhere else to live, Adesola said that, ‘it is best not to complain’, as he did not want to ‘cause problems or irritate anyone’. At assessment, he said that he remembered being with his ‘real parents’ at the time of his birth and early years, and that those he now refers to as his parents are in fact imposters. Adesola had contacted the police several times about this and believed them to be investigating the issue.

Adesola said that he had first begun hearing voices during the Christmas break of his first term at university, where he was studying for an IT-based degree. He said that he had been struggling with the demands of the degree and had been pressured by his parents to pursue this qualification. He had instead wanted to study Performing Arts, as he had a passion for music. Perhaps significantly, Adesola’s parents had recently returned from a West African country just before he had begun to hear voices. He said that, soon after he had begun to hear voices, his parents took him to this West African country for an extended period. Adesola remained concerned thereafter that he would be taken to West Africa again, reporting that his parents had told him they would do so ‘if his situation did not improve’. He was strongly against going to West Africa again, as he felt that the standard of living was worse, relative to the United Kingdom. Importantly, Adesola also reported having experienced ‘bad side-effects’ as a result of the medication that he had been prescribed in West Africa following his first experience of psychosis.

V. FORMULATION

Adesola's case was formulated as follows, based on the cognitive model of the positive symptoms of psychosis [6] (see Figure 1).

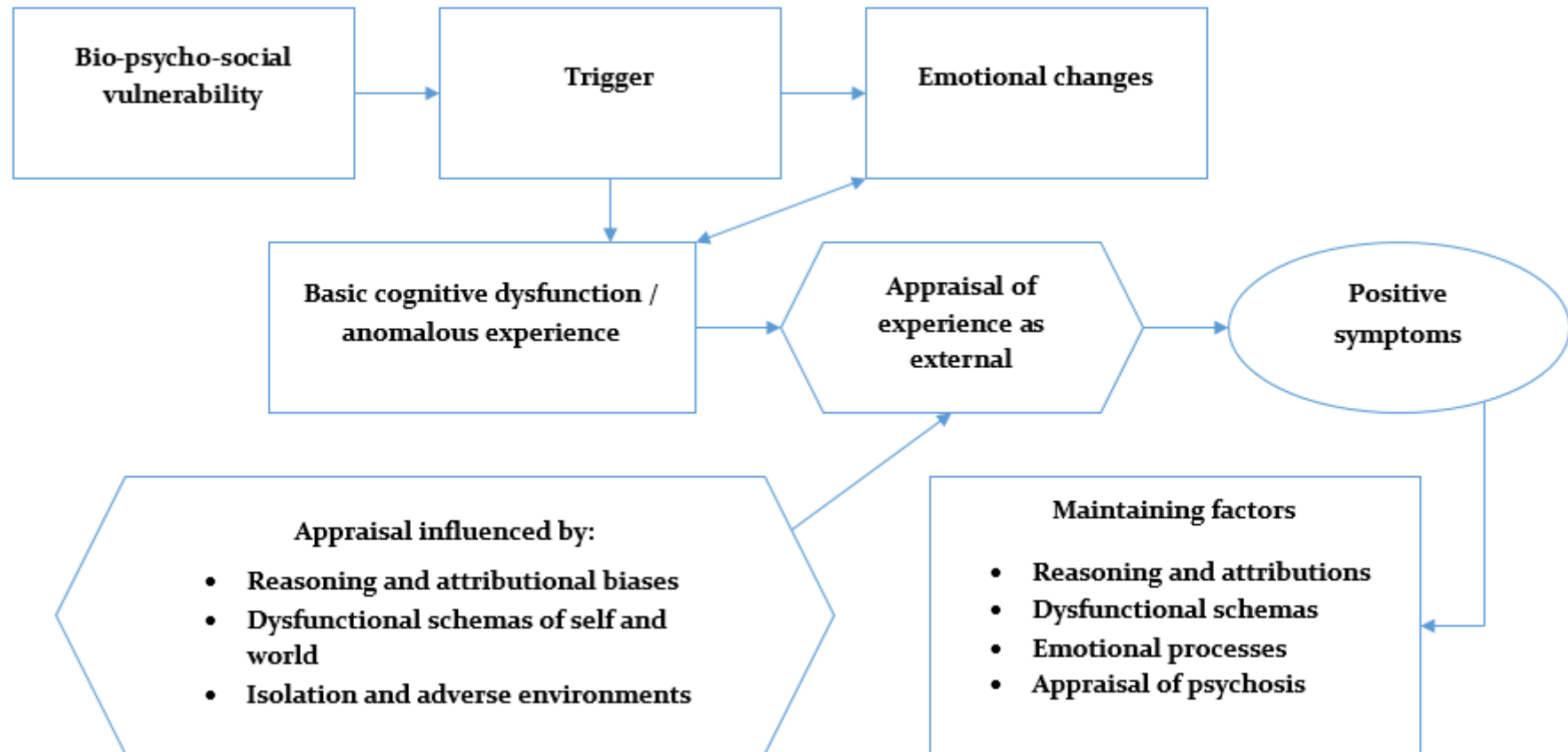


Figure 1: A model of the positive symptoms of psychosis [based on the cognitive model developed by 6]

According to Garety *et al.* [6], Adesola would have had an underlying biopsychosocial vulnerability/predisposition to develop the positive symptoms of psychosis. The model proposes that such symptoms most frequently result from a triggering event (e.g., life-events, adverse environments, illicit drug use and/or periods of isolation) that brings about a disruption of cognitive processes [28], leading to anomalous conscious experiences (e.g., thoughts appearing to be broadcast, thoughts experienced as voices, unconnected events seeming to be linked). The triggering event(s) (i.e. the point at which he began to hear voices) for Adesola seems to have been the stress of his university course, together, possibly, with his family's returning from West Africa.

These anomalous experiences often feel external and understandably threatening, triggering emotional arousal and a 'search for explanation' [29]. Biased conscious appraisal processes (e.g., jumping to conclusions, an externalising attributional bias and social cognition deficits) are said to be crucial at this point, facilitating a conclusion that the distressing experiences are in fact externally caused [30]. Although, given that I saw Adesola some years after these earlier experiences and it would thus not have been possible to specify the nature of any initially biased appraisal processes, it was clear that he had settled upon an external attribution for his anomalous experiences (e.g., 'they are the voices of everyone in the world', 'spirits' and 'yobs trapped in purgatory', etc.). The model posits that social isolation contributes to the acceptance of the psychotic appraisal by reducing access to alternative, more normalising explanations [31]. This certainly seemed relevant to Adesola, who seemed to have been a somewhat isolated figure throughout his life.

Dysfunctional schemas and adverse environments are an important aspect of the cognitive model [e.g., 32, 33]. For example, Close and Garety [34] found that hallucinations and delusions that have negative content are associated with negative self-concepts. Garety *et al.* [6] cite research of low self-esteem developing in specific social contexts [35], such as living with unsupportive, high expressed-emotion families [e.g., 36]. The authors contend that psychotic beliefs may be more firmly held if consistent with firmly-held, distorted beliefs concerning the self (e.g., that one is bad), others (e.g., that others are hostile) and the world (e.g., that the world is dangerous). Adesola certainly seemed to have a low self-concept, as manifested, for example, in his stating that his confidence 'vanishes' in the presence of other people. It seems reasonable to speculate that the difficult relationships he reported with his family

might have been significant: Adesola's reports of these interactions (which he described as 'stressful') seemed to be suggestive of significant levels of expressed emotion (e.g., refusing to accept his mental health difficulties and pressuring him to get a job). Furthermore, his mother's apparent insistence that he get a job would likely have acted as an ongoing reminder of his difficulties (e.g., his daily experiences of chronic anxiety and feeling unable to leave his home), serving to maintain and reinforce his seemingly poor self-concept. It is not difficult to imagine that his traumatic-sounding experiences of being taken to Wes Africa, against his will (during which he experienced 'bad side-effects' of medication), following his first psychotic episode, might have led to a distrust of others and perception of the world as being unpredictable and dangerous. Strikingly, Mueser *et al.* [37] have reported a 98 per cent lifetime prevalence of traumatic events in those with psychosis.

Garety *et al.* [6] also draw attention to the potential importance of the wider social environment, citing evidence of greater incidence of psychosis in inner-cities and in certain ethnic groups [38, 39]. These authors contend that the social marginalisation associated with such factors may also contribute to the development of negative schemas, often involving social humiliation and subordination [40]. Given this, it may be relevant that Adesola was a Black man who grew-up and lived in South-East London.

It is notable, given the foregoing, that both Adesola's experiences of command hallucinations (by the spirits) and his relationships with family members were, to a large extent, characterised by passivity. Thus, his appraisal that he needed to be compliant and obedient to the voices' instructions seemed to echo the rules he had for interactions with his family (e.g., 'it is best not to complain'). It seems reasonable to speculate that this passive approach towards social interactions may have been established during childhood and adolescence (e.g., as a result of unsuccessful attempts to challenge his parents).

Adesola's delusions of being Jesus Christ, with 'magical powers' on a mission to fulfil his destiny and 'save souls in purgatory', was conceptualised as a grandiose defence against his seemingly low self-concept [e.g., 41]. In perceiving himself (though not necessarily consciously) as the saviour of humankind, he was an all-powerful figure, able to achieve anything. This contrasted sharply with his real-world circumstances, in which he was socially isolated, financially dependent and struggled to tolerate the anxiety of being out in public. His ideas around being all-powerful may also have served

as a psychological escape from the powerlessness and associated emotions he seemed to experience in relation to his mother and other family members, whom he reported threatened to remove him to West Africa if ‘his situation did not improve’.

The origins of Adesola’s fear of tall towers was challenging to formulate. It is possible that this represented a form of ‘reassuring’ inversion of his anxieties around being vulnerable whilst out in public in general. Thus, due to a combination of neuropsychological vulnerability, past experiences, his delusional belief system and symptoms of social anxiety, he was fearful of being attacked whilst outside his home, which was understandably a significant source of distress. If, however, his mere presence close to tall towers could bring about their collapse and destruction, then this would mean that he himself had significant power(s) – that he was potentially dangerous to others – and may, on some level, have served to act against his feelings of vulnerability. Regardless of its origins, his subsequent avoidance of and escape from being in close proximity to tall towers served to maintain these fears via his preventing the receipt of disconfirmatory evidence [42]. In addition, an information-processing bias in which he spent time gathering and focusing on information related to the dangers of tall towers (e.g., building-collapses in other parts of the world) was also significant in providing further ‘evidence’ for his fears [42]. The relationship between Adesola’s positive symptoms and his anxiety seemed to be bi-directional, such that his positive symptoms (e.g., the loudness and emotional tone of the voices, according to his verbal self-report) became more salient and intense when close to tall towers (and when in public, generally) and *vice versa*.

VI. AIMS OF THE INTERVENTION

Adesola’s specific aim was to reduce his anxiety around buildings collapsing in his proximity due to his powers, such that he would feel able to regularly travel to and from various day/community centres and support groups around South-East London – which his anxiety had been preventing him from doing. At the beginning of psychology sessions, Adesola was concerned that, by talking about his concerns, he would be ‘exposed’ – by which he meant that the voices on the airwaves would become aware that he was talking about them (and may take retaliatory action). However, he also displayed some ambivalence in his attitude towards the voices, stating that he wanted to have more freedom and to go out more, in particular to meet friends. He also said that he felt alienated from others and that he wanted to find employment and

live a more meaningful life. Fowler *et al.* [16] recommend working towards a specific, valued goal as a central part of CBTp.

VII. INTERVENTION

A modified form of CBT, tailored to Adesola's presentation, was delivered. The intervention consisted of outpatient sessions, focusing principally on Adesola's fear of tall towers. In accordance with the guidance provided by Fowler *et al.*'s treatment manual [16], the intervention was, in various ways, delivered more flexibly than in non-psychosis populations. Thus, for example, the length of each session varied, depending on the extent to which Adesola felt able to continue, although, on average, sessions lasted around 45 minutes. The intervention was delivered by both myself and my supervisor. The first six sessions were focused on an extended assessment and the development of a shared formulation of Adesola's difficulties, as well as building rapport and engagement with him.

In order to achieve Adesola's main goal of being able to travel around South-East London independently, the intervention primarily sought to challenge and restructure his fear of tall towers in a non-threatening, compassionate yet evidence-based manner. Thus, we did not seek to challenge his beliefs regarding his being Jesus Christ in communication with spirits who were trying to save souls from purgatory, etc. Instead, we focused on contextualising the spirits' fears of harm coming to Adesola and/or others due to tall towers collapsing as their making predictions with the best intentions (i.e. to keep Adesola and others safe), but that, without testing-out these ideas/fears, it would not be possible to know whether they were accurate or not. This provided a rationale for empirically exploring them – that is, for Adesola to expose himself to tall towers (e.g., walking towards and standing in front of them) to both explore whether the fears were proportional and also to learn to tolerate the associated anxiety. We developed a hierarchy of feared tower-related scenarios and, across the sessions, worked 'up' these scenarios at a pace with which Adesola felt comfortable.

Each of the 'steps' on this hierarchy involved our walking, each week, progressively closer to each of two tall towers close to the team base, which Adesola had respectively named 'Bosling' and 'Champion'. Subjective units of distress (i.e. 0-10) were taken prior to, during and after Adesola's having been gently encouraged to walk towards the towers. Following received wisdom on CBTp, the exposure sessions were conducted

more flexibly than would typically be the case in non-psychosis populations. Thus, on some occasions, Adesola's anxiety during exposures seemed to be increasing/to have increased to a level that was leading him to experience more florid positive symptoms (e.g., the spirits becoming louder and/or more threatening) and, understandably, elevated his level of distress. We thus regularly 'checked-in' with Adesola regarding what the spirits were saying and sometimes left the situation so as to reduce the chances of Adesola finding the sessions too aversive. At such times, we came back to the CMHT in order to debrief and allow a period for his anxiety to reduce closer to baseline.

In tandem with the exposure tasks, we also spent time thinking about the evidence for/against and likelihood of large buildings in London collapsing. For example, we wrote a letter to the office of the then-mayor of London, Boris Johnson, stating our concern about the safety of tall towers in the city and asking for information regarding measures used to ensure public safety (to which we received a reply, including directions to a number of webpages linked to the Mayor's Office website outlining such safety procedures). We also searched the Internet and read webpages (some of which referred to in the Mayor's reply) concerning the many and varied safety and architectural procedures involved in constructing a large building in London. Prior to and following such tasks, Adesola was asked to rate his concern (from 0 to 10) regarding the safety of tall towers. Adesola found some of this information compelling and we often referred back to it during subsequent exposure tasks.

VIII. MEASURES

Quantitative outcome(s):

The Clinical Outcomes in Routine Evaluation – 10-item version [CORE-10; 43] was administered at pre-, mid- and post-sessions as a quantitative outcome measure. The CORE-10 is a widely-administered, 10-item self-report scale comprising questions across a range of domains, including symptoms of depression, anxiety, general functioning, social relationships and close relationships, trauma, physical symptoms and risk. It is an abbreviated version of the Clinical Outcomes in Routine Evaluation-Outcome Measure [CORE-OM; 44], with each item being rated on a 5-point Likert scale, ranging from 0 ('not at all') through to 4 ('most or all of the time'). Items can be summed to produce an overall score from 0-40, with greater scores indicating greater

levels of distress. Barkham *et al.* [43] suggest a cut-off point of 13 and above for indicating depression and a score of 11 and above for general psychological distress, and report an α -reliability of 0.90. At the beginning of the intervention, Adesola had a score of 18, above threshold for depression and psychological distress.

In addition to the overall score, given that the CORE-10 includes items across a range of symptom- and functioning-domains, Adesola's score for each of the ten items was also explored across the three administration-points. This was done in order to explore, more specifically, his particular strengths and challenges. His responses at baseline are presented in Table 1.

Table 1: Adesola's responses (and scores) to each item of the CORE-10 at the beginning of sessions

Item	Adesola's response (and score)
1. I have felt tense, anxious or nervous	Most or all of the time (4)
2. I have felt I have someone to turn to for support when needed*	Often (1)
3. I have felt able to cope when things go wrong*	Most or all of the time (0)
4. Talking to people has felt too much for me	Only occasionally (1)
5. I have felt panic or terror	Often (3)
6. I have made plans to end my life	Not at all (0)
7. I have had difficulty getting to or staying asleep	Often (3)
8. I have felt despairing or hopeless	Often (3)
9. I have felt unhappy	Sometimes (2)
10. Unwanted images or memories have been distressing me	Only occasionally (1)
Total score = 18	

Note: Items rated on the following 5-point Likert scale: 0 = 'not at all', 1 = 'only occasionally', 2 = 'sometimes', 3 = 'often' and 4 = 'most or all of the time'.

* Item reverse-scored.

From the table, it can be seen that, in accordance with the difficulties for which he was seeking treatment, Adesola's highest-scoring responses were mainly related to issues of anxiety.

We were mindful not to ask Adesola not to complete more outcome measures, so as not to make the process too arduous for him.

Qualitative outcome(s):

We collaboratively developed a SMART (Specific, Measureable, Achievable, Relevant and Time-based) goal of Adesola's being able to travel to and from day/community centres around South-East London independently by the end of the intervention.

IX. DIFFICULTIES/CHALLENGES

The complexities of Adesola's presentation contributed to some challenges throughout the intervention. He often either cancelled sessions or did not attend (DNA'd) them, thereby reducing the amount of therapeutic time. After such occasions, Adesola tended to report that the spirits had been 'speaking very loudly', preventing him from going out.

Unfortunately, there was a period around the mid-point of sessions when Adesola and I did not have a session for almost a month, due to his experiencing a crisis of severe anxiety. Conducting an emergency assessment, the Home Treatment Team (HTT) found him acutely unwell, telling them that he was unable to speak and that he was only able to communicate in writing. Adesola wanted verification that he was Jesus and the next world leader and, 'for the relay that has voices and the birds' singing to be less'. He said that he could not leave the house due to 'everybody staring at him and being able to read his thoughts'. It emerged that Adesola had bought a box of weight-loss pills from the Internet, taking 14 of them in one day. These consisted almost entirely of caffeine and seem to have acted as a physiological trigger to his crisis. As well as increasing his anxiety, this had, for several days, made him feel dizzy, shaky and caused him to vomit a number of times. Believing these symptoms related to his anti-psychotic medication, Adesola had thrown his entire prescription out, his abrupt cessation of medication likely to have maintained his crisis during these weeks.

Adesola found it difficult to report/discuss his anxiety, instead often projecting an apparently superficial positivity. For example, when asked how he was feeling during

exposure tasks, he tended to report ‘feeling perfectly fine’, despite seeming rather anxious. This was formulated as a manifestation of his ongoing desire ‘not to cause any trouble’ and seemed to stem in part from his misperceiving that he was expected *not* to feel anxiety during the tasks (and that doing so would indicate failure/his causing trouble). Despite our regularly outlining the purpose of the exposures (i.e. to gradually reduce his high anxiety), as well as encouraging him to report his anxiety, Adesola continued to find this difficult. Though we tried to discern his anxiety-level via his demeanour, it was often nevertheless difficult to tell what his anxiety was at any given moment, posing obvious challenges during the tasks.

Adesola’s ongoing concerns about being taken to West Africa again periodically increased at times when his mother returned to London (from West Africa). Although we did not discuss this (as well as his relationship with his parents more broadly) in detail during my sessions with him, it seems likely that the associated stress served to increase his background/baseline levels of anxiety, possibly making the exposure tasks (even) more challenging for him.

At times, Adesola seemed ambiguous about fully engaging due to concerns that he would be made to find employment. We sought to reassure him by saying we felt it would be difficult for him to work at present and that he would be supported by the team should he be called for a workplace assessment.

X. RESULTS

Quantitative outcomes:

Adesola’s self-reported scores on each item of the CORE-10 at baseline, mid-sessions and the end of sessions are shown in Table 2.

Table 2: Adesola's responses (and scores) to each item of the CORE-10 at baseline, mid-sessions and at the end of sessions

Item	Baseline	Mid-sessions	End of sessions
1. I have felt tense, anxious or nervous	Most or all of the time (4)	Only occasionally (1)	Sometimes (2)
2. I have felt I have someone to turn to for support when needed*	Often (1)	Often (1)	Often (1)
3. I have felt able to cope when things go wrong*	Most or all of the time (0)	Sometimes (2)	Sometimes (2)
4. Talking to people has felt too much for me	Only occasionally (1)	Not at all (0)	Not at all (0)
5. I have felt panic or terror	Often (3)	Sometimes (2)	Sometimes (2)
6. I have made plans to end my life	Not at all (0)	Not at all (0)	Not at all (0)
7. I have had difficulty getting to or staying asleep	Often (3)	Sometimes (2)	Only occasionally (1)
8. I have felt despairing or hopeless	Often (3)	Sometimes (2)	Sometimes (2)
9. I have felt unhappy	Sometimes (2)	Only occasionally (1)	Sometimes (2)
10. Unwanted images or memories have been distressing me	Only occasionally (1)	Only occasionally (1)	Not at all (0)
Total score = 18 Total score = 12 Total score = 12			

Note: Items rated on the following 5-point Likert scale: 0 = 'not at all', 1 = 'only occasionally', 2 = 'sometimes', 3 = 'often' and 4 = 'most or all of the time'.

* Item reverse-scored.

It can be seen from the table that, across the intervention, there was a clear reduction in Adesola's overall CORE-10 scores, such that his baseline score of 18 – that is, above threshold for depression – had, by the mid-point in sessions, reduced to 12 – that is,

below threshold for depression – though this score remained above the cut-off for general psychological distress. This reduction was maintained to the end of sessions. Notably, 50 percent of the reduction in his overall score (i.e. 3) was attributable to those items relating more specifically to anxiety (i.e. 1 and 5) – that is, those aspects that were being specifically targeted as part of the intervention.

In terms of his SMART goal, although Adesola was not able to travel *independently* around South-East London by the end of the intervention, he was able to tolerate travelling to an area with two day/community centres (that he was strongly invested in attending) in the company of someone else (e.g., his Support Worker). Although he continued to find being close to tall buildings understandably anxiety-provoking (given the long period of previous avoidance), he was better able to approach these feared stimuli and tolerate the anxiety without escaping the situation. These clinical observations are consistent with his reduced anxiety ratings on CORE-10 items. In a session towards the end, Adesola himself stated that he had ‘greater mental strength to get close to tall towers’.

XI. DISCUSSION

The modified CBTp via graded exposure course was moderately successful in facilitating Adesola’s goal of being able to travel to and from day centres around South-East London. He had previously felt himself unable to travel to certain areas (regardless of being accompanied or not), due to a large number of tall towers (i.e. flats and office-blocks) in the vicinity and so this represented a significant achievement. It seems likely that Adesola’s progress may have been hindered somewhat by the crisis he experienced involving the slimming-pills. At the same time, that he was nevertheless able to make some progress in the context of this crisis, as well as other ongoing stressors (e.g., the relationship with his family), should be borne in mind. Adesola continues to work on his fears around tall towers with my supervisor from this placement, and also now spends one day a week travelling to and spending time at his day centres with his Support Worker as additionally work towards his goal. Thus, our sessions might be viewed as the initial steps towards (hopefully) further progress in future.

Overall, after working with Adesola, the logic behind Fowler *et al.*’s [16] recommendation of working towards a specific and valued goal for the patient in CBTp was reinforced for me. The complex difficulties related to psychosis, not only due to

personal symptoms and other comorbidities, but also related to (lack of) employment, financial difficulties and general social marginalisation [45, 46], mean that those with the diagnosis can often struggle to engage and maintain engagement in therapy (e.g., due to associated distress). For Adesola, due to such issues as his ongoing concern around being removed to West Africa, it may not have been practical, at that point in his recovery, for us to have worked on other, perhaps larger issues (e.g., the way he perceived/relationship that he had with his auditory hallucinations, relations with family members).

At the same time, that the intervention focused so specifically on his (intra-individual) fear of tall towers might be considered a limitation. After all, his difficult relationships with family members was formulated as having possibly had a significantly detrimental impact on his self-esteem, as well as being related to the content of and way he interacted with his auditory hallucinations. Thus, in order to address these issues, since my work with Adesola ended, he has been referred, and is currently on the waiting-list for, family therapy for psychosis. His mother has agreed to attend these sessions with him. He said that he wanted their relationship to be 'less fighty'.

XII. REFLECTIONS

Working with Adesola led me to reflect on a range of factors relating to diversity and cultural differences, which I believe have broadened my awareness of these issues. Throughout our work together, I experienced particularly high levels of sympathy; at times, it seemed that I almost felt guilty. Reflecting on these feelings as part of supervision, I believe that they were related to Adesola and I's both being male and having been born in the same year, yet our lives having taken different paths. I reflected on Adesola and his family's being Black and his having grown-up in a relatively deprived part of the country (i.e. South-East London). I wondered whether and to what extent these factors may have been important in his parents' pressuring him (as Adesola reported) into studying for a degree high in prestige and a first step in a potentially lucrative career (i.e. Software Engineering), but which he had not wanted to do and had seemingly stretched his mental resources, putting him under significant stress (which seemed to act as a trigger for his first psychotic experience and his seemingly traumatic temporary removal to West Africa). Overall, working with Adesola led me to reflect on the many life experiences, particularly when we are younger, that operate on us and over which we often have little control – but which

have the potential to significantly influence how we view and experience the world – and how we see ourselves within it.

REFERENCES

1. Association, A.P., *Diagnostic and statistical manual of mental disorders (DSM-5®)*. 2013: American Psychiatric Pub.
2. Heinrichs, R.W., et al., *Cognition as a central illness feature in schizophrenia*, in *Cognitive Impairment in Schizophrenia: Characteristics, Assessment and Treatment*, P.D. Harvey, Editor. 2013, Cambridge University Press: The Edinburgh Building, Cambridge.
3. Waller, H., et al., *Low intensity cognitive behavioural therapy for psychosis: a pilot study*. *Journal of behavior therapy and experimental psychiatry*, 2013. **44**(1): p. 98-104.
4. Stafford, M.R., et al., *Early interventions to prevent psychosis: systematic review and meta-analysis*. *Bmj*, 2013. **346**: p. f185.
5. Slade, M., *Personal recovery and mental illness: A guide for mental health professionals*. 2009: Cambridge University Press.
6. Garety, P.A., et al., *A cognitive model of the positive symptoms of psychosis*. *Psychological medicine*, 2001. **31**(02): p. 189-195.
7. Johnstone, E.C., *Disabilities and circumstances of schizophrenic patients: A follow-up study*. *The British Journal of Psychiatry*, 1991.
8. Palmer, B.A., V.S. Pankratz, and J.M. Bostwick, *The lifetime risk of suicide in schizophrenia: a reexamination*. *Archives of general psychiatry*, 2005. **62**(3): p. 247-253.
9. Tarrier, N., et al., *Suicide behaviour over 18 months in recent onset schizophrenic patients: the effects of CBT*. *Schizophrenia research*, 2006. **83**(1): p. 15-27.
10. Achim, A.M., et al., *How prevalent are anxiety disorders in schizophrenia? A meta-analysis and critical review on a significant association*. *Schizophrenia bulletin*, 2011. **37**(4): p. 811-821.
11. Buckley, P.F., et al., *Psychiatric comorbidities and schizophrenia*. *Schizophrenia bulletin*, 2009. **35**(2): p. 383-402.

12. Freeman, D. and P.A. Garety, *Connecting neurosis and psychosis: the direct influence of emotion on delusions and hallucinations*. Behaviour research and therapy, 2003. **41**(8): p. 923-947.
13. Birchwood, M., et al., *Social anxiety and the shame of psychosis: a study in first episode psychosis*. Behav Res Ther, 2007. **45**(5): p. 1025-37.
14. Fowler, D., R. Rollinson, and P. French, *Adherence and competence assessment in studies of CBT for psychosis: current status and future directions*. Epidemiology and Psychiatric Sciences, 2011. **20**(02): p. 121-126.
15. Wing, J.K., *Schizophrenia*, in *Theory and Practice of Psychiatric Rehabilitation*, F.N. Watts and D.H. Bennett, Editors. 1983, Wiley: Chichester.
16. Fowler, D., P. Garety, and E. Kuipers, *Cognitive behaviour therapy for psychosis: Theory and practice*. Vol. 25. 1995: Wiley.
17. Kuipers, E., et al., *Cognitive, emotional, and social processes in psychosis: refining cognitive behavioral therapy for persistent positive symptoms*. Schizophrenia Bulletin, 2006. **32**(suppl 1): p. S24-S31.
18. Peters, E., et al., *Delusional ideation in religious and psychotic populations*. British Journal of Clinical Psychology, 1999. **38**(1): p. 83-96.
19. Smith, B., et al., *Emotion and psychosis: links between depression, self-esteem, negative schematic beliefs and delusions and hallucinations*. Schizophrenia research, 2006. **86**(1): p. 181-188.
20. Peters, E., et al., *A randomised controlled trial of cognitive behaviour therapy for psychosis in a routine clinical service*. Acta Psychiatrica Scandinavica, 2010. **122**(4): p. 302-318.
21. NICE, *Schizophrenia. Core interventions in the treatment and management of schizophrenia in primary and secondary care (update)*. 2009, National Institute for Health and Clinical Excellence: London.
22. Freeman, D., R.P. Bentall, and P. Garety, *Persecutory delusions: Assessment, theory and treatment*. 2008, Oxford: Oxford University Press.
23. Pilling, S., et al., *Psychological treatments in schizophrenia: I. Meta-analysis of family intervention and cognitive behaviour therapy*. Psychological medicine, 2002. **32**(05): p. 763-782.

24. Tarrier, N. and T. Wykes, *Is there evidence that cognitive behaviour therapy is an effective treatment for schizophrenia? A cautious or cautionary tale?* Behaviour research and therapy, 2004. **42**(12): p. 1377-1401.
25. Zimmermann, G., et al., *The effect of cognitive behavioral treatment on the positive symptoms of schizophrenia spectrum disorders: a meta-analysis.* Schizophrenia research, 2005. **77**(1): p. 1-9.
26. Cormac, I., et al., *Cognitive behaviour therapy for schizophrenia.* The Cochrane Library, 2002.
27. Excellence), N.N.I.f.H.a.C. *Psychosis and schizophrenia in adults: prevention and management. Clinical guideline (CG178).* 2014.
28. Garety, P.A. and D.R. Hemsley, *Delusions: Investigations into the psychology of delusional reasoning.* Vol. 36. 1997: Psychology Press.
29. Maher, B.A., *Anomalous experience and delusional thinking: The logic of explanations.* 1988: John Wiley & Sons.
30. Garety, P.A. and D. Freeman, *Cognitive approaches to delusions: a critical review of theories and evidence.* British journal of clinical psychology, 1999. **38**(2): p. 113-154.
31. White, R., et al., *The social context of insight in schizophrenia.* Social psychiatry and psychiatric epidemiology, 2000. **35**(11): p. 500-507.
32. Trower, P. and P. Chadwick, *Pathways to defense of the self: A theory of two types of paranoia.* Clinical psychology: Science and practice, 1995. **2**(3): p. 263-278.
33. Kinderman, P. and R.P. Bentall, *Self-discrepancies and persecutory delusions: evidence for a model of paranoid ideation.* Journal of abnormal psychology, 1996. **105**(1): p. 106.
34. Close, H. and P. Garety, *Cognitive assessment of voices: further developments in understanding the emotional impact of voices.* British Journal of Clinical Psychology, 1998. **37**(2): p. 173-188.
35. Brown, G., et al., *Self-esteem and depression: II. Social correlates of self-esteem.* Social Psychiatry and Psychiatric Epidemiology, 1990.

36. Bebbington, P. and L. Kuipers, *The predictive utility of expressed emotion in schizophrenia: an aggregate analysis*. Psychological medicine, 1994. **24**(03): p. 707-718.
37. Mueser, K.T., et al., *Trauma and posttraumatic stress disorder in severe mental illness*. Journal of consulting and clinical psychology, 1998. **66**(3): p. 493.
38. Bhugra, D., et al., *Incidence and outcome of schizophrenia in whites, African-Caribbeans and Asians in London*. Psychological medicine, 1997. **27**(04): p. 791-798.
39. Mortensen, P.B., et al., *Effects of family history and place and season of birth on the risk of schizophrenia*. New England Journal of Medicine, 1999. **340**(8): p. 603-608.
40. Birchwood, M., et al., *The power and omnipotence of voices: subordination and entrapment by voices and significant others*. Psychological medicine, 2000. **30**(02): p. 337-344.
41. Neale, J.M., *Defensive functions of manic episodes*. 1988.
42. Freeman, D. and P.A. Garety, *Cognitive therapy for an individual with a long-standing persecutory delusion*, in *A Casebook of Cognitive Therapy for Psychosis*. Hove: Brunner-Routledge, A.P. Morrison, Editor. 2002. p. 173-196.
43. Barkham, M., et al., *The CORE-10: A short measure of psychological distress for routine use in the psychological therapies*. Counselling and Psychotherapy Research, 2013. **13**(1): p. 3-13.
44. Barkham, M., et al., *Suitability and utility of the CORE-OM and CORE-A for assessing severity of presenting problems in psychological therapy services based in primary and secondary care settings*. The British Journal of Psychiatry, 2005. **186**(3): p. 239-246.
45. Marwaha, S. and S. Johnson, *Schizophrenia and employment*. Social psychiatry and psychiatric epidemiology, 2004. **39**(5): p. 337-349.
46. Marwaha, S., et al., *Rates and correlates of employment in people with schizophrenia in the UK, France and Germany*. The British Journal of Psychiatry, 2007. **191**(1): p. 30-37.

CLINICAL CASE STUDY 3

*‘THERE WILL BE BLOOD- (AND INJECTION-RELATED
STIMULI)’*

COGNITIVE-BEHAVIOURAL THERAPY VIA GRADED
EXPOSURE FOR BLOOD-INJECTION-INJURY PHOBIA WITH
A 13-YEAR-OLD FEMALE

SUPERVISED BY DR. LAUREN PEILE

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I. LITERATURE REVIEW

I.1) BLOOD-INJECTION-INJURY PHOBIA: DEFINITION, CHARACTERISTICS, EPIDEMIOLOGY AND TREATMENT

Blood-Injection-Injury (BII) phobia refers to a condition in which individuals display intense, irrationally-high levels of fear and anxiety in response to real or imagined exposures to blood, physical injury, injections or other medically-related procedures [1]. BII phobia is relatively common, with an estimated prevalence-rate of between 3 to 4 per cent in the general population [2]. A commonly-observed and unfortunate consequence of BII phobia is an avoidance not only of blood and injections in particular, but also of medically-related stimuli and scenarios more broadly. As a result, individuals tend not to report to / refuse offers of appointments at clinics and hospitals. As well as serving to maintain and possibly deepen their anxieties, such avoidance behaviour increases the risks to individuals' physical health, as they miss a range of medical procedures, from routine check-ups through to surgical operations [3]. Furthermore, BII phobia can also restrict potential occupational choices, by excluding employment in medical and related contexts, as well as limiting life-decisions more broadly, such as travel to countries for which vaccinations are recommended or required. Regarding society more broadly, BII phobia also reduces the numbers of people donating blood [4].

In virtually all forms of specific phobia, exposure to feared stimuli precipitates a common physiological response, involving increased blood-pressure and an elevated heart-rate (i.e. tachycardia). BII phobia, however, is unique, in that, upon real or imagined exposure to blood-, injection- and/or related stimuli, individuals typically experience a diphasic cardiovascular response, whereby an initial increase in blood-pressure and tachycardia is followed by bradycardia (i.e. abnormally slow heartbeat), diaphoresis (i.e. sweating), hypotension, shock, vertigo, nausea and, rarely, asystole (i.e. ceasing of heartbeat) and death [5, 6]. The latter part of this physiological response, termed 'vasovagal syncope', often leads to fainting reactions in the presence of feared stimuli [7]. It is estimated that, in around 80 per cent of those with BII phobia, the phobic response is characterised by syncope or *pre-syncope* [that is, light-headedness, muscular weakness, blurred vision and feeling faint - as opposed to syncope, in which the individual does in fact faint; 8] [9, 10]. Recently, disgust-sensitivity – that is, 'the degree to which a person feels disgusted in response to a variety of stimuli' [11, p.54] –

has been posited as an important element in BII [12]. It appears that high disgust-sensitivity appears to elicit parasympathetic activity and reductions in blood-pressure in BII phobics [13], thereby making an individual more susceptible to fainting [14, 15].

Given the potentially serious health consequences of BII phobia, it is important that individuals are provided with effective treatment. The existing evidence-base indicates the most efficacious treatment to be Cognitive-Behavioural Therapy (CBT) based on graded exposure [e.g., 16, 17-19]. Although there do not appear to be any NICE guidelines for BII phobia, CBT would appear to be an effective first-line approach. Given the characteristic vasovagal response in BII phobia, Mednick and Claar [20] recommend that applied muscle-tension (AMT) is incorporated into CBT interventions (prior to beginning graded exposure) – the aim of AMT being to counteract the syncopal response on exposure to feared stimuli, such that the patient is able to experience habituation [18]. Chapman and DeLapp [3] have recently described the successful application of manualised CBT, incorporating applied muscle-tension, in a single case design involving an adult male with longstanding BII phobia.

I.II) OBSESSIVE-COMPULSIVE DISORDER: CHARACTERISTICS, EPIDEMIOLOGY AND TREATMENT

Obsessive-Compulsive Disorder (OCD) is characterised by the presence of obsessions and/or compulsions [1]. Obsessions can be defined as recurrent and persistent thoughts (e.g., of contamination), urges (e.g., of violent or horrific scenes) or images (e.g., to stab someone) that are experienced by the individual as intrusive and unwanted, causing marked distress. Compulsions, on the other hand, are repetitive behaviours (e.g., washing, checking) or mental rituals (e.g., counting, repeating words silently) that the individual feels must be performed in response to an obsessive thought or according to rules that the individual believes must be rigidly adhered to. Most OCD patients have both obsessions and compulsions. By performing compulsions, the patient hopes to reduce the distress engendered by obsessions or to prevent a feared event (e.g., becoming unwell). However, the compulsions are either not tangibly related to the feared event (e.g., fixing one's gaze on a certain part of the room in order maintain one's sanity) or are obviously excessive (e.g., washing one's hands 50 times a day). The obsessions and/or compulsions are time-consuming and cause clinically-significant distress and/or functional impairment in social, occupational or other life areas. OCD

can follow a chronic course if left untreated [21]. The 12-month prevalence of OCD across cultures is estimated at between 1.1-1.8% [1].

CBT is known to be the most effective intervention for OCD, in combination with a selective serotonin reuptake inhibitor (SSRI) medication [22, 23]. A fundamental aspect of CBT for OCD is Exposure and Response-Prevention (E/RP), which involves the patient repeatedly and systematically exposing themselves to the feared event(s) associated with their obsession(s), whilst simultaneously resisting the urge to engage in their compulsion(s).

II. THE CLIENT

The client – ‘Sarah’ (not her real name) – was a 13-year-old White British female. She was an only-child of above-average academic ability (she was in the top sets for all subjects at an independent girls’ school).

III. PRESENTING PROBLEM

Sarah was referred into the service by her Consultant Paediatric Nephrologist¹ in October 2014. It was reported that Sarah felt faint and unwell when blood was discussed or if she was exposed to a cue related to blood or blood-tests (e.g., a blood-pressure cuff or being asked to hold-out her arm prior to a needle being inserted). This had led her to avoid certain important immunisations (e.g., the MMR and HPV vaccines) and blood-tests. The latter was particularly significant because, in 2005, when she was 4 years old, Sarah had had an episode of haemolytic uraemic syndrome (HUS). HUS is predominantly observed in children and is characterised by anaemia (caused by the destruction of red blood cells), low platelet count and acute kidney failure [24]. Sarah’s HUS had been treated during a month-long hospitalisation, wherein she underwent a 2-week period of inpatient dialysis. She was considered to have made a good recovery and to be in good physical health. It was nevertheless necessary for her to undergo regular blood-tests thereafter, in order to monitor her red blood cell- and platelet-counts and thereby check for signs of potential relapse. Sarah’s avoidance of these blood-tests therefore represented an ongoing risk to her physical health.

¹ A medical doctor specialising in disorders of the kidneys.

Her anxiety meant that she was also avoiding watching television programmes or films that may contain images of injections, injuries, operations or any other blood-related contexts; this placed restrictions on Sarah's ability to socialise (e.g., spending time with family via watching television in the living-room, going to the cinema with friends, etc.). In addition, at school, she would often feel compelled to leave the classroom during Biology lessons concerning the circulatory system, the workings of internal organs, etc., due to feelings of nausea and her fear of fainting. Overall, therefore, the client's BII phobia was significantly compromising her ability to function and to fulfil role-expectations across various life-contexts.

IV. BACKGROUND

Sarah had had a previous diagnosis of OCD and had been treated previously (in 2011, when she was 10), via CBT using Exposure with Response-Prevention (E/RP), in the same team in which the treatment detailed in the present case study took place. At assessment, her OCD was of moderate severity (she scored '20' on the Children's Yale-Brown Obsessive-Compulsive Scale, or CY-BOCS). Sarah's ego-dystonic obsessive thoughts centred around her mother being 'fat', 'old' and 'wrinkly'. Sarah felt compelled to share these obsessive thoughts with her mother. Sarah also had intrusive thoughts about her mother having a disease or being stabbed and dying and, consequently, repeatedly sought assurance, from her mother, that she was safe and well. In addition, the client felt anxious that she might have sworn or made an accidental, offensive gesture and compulsively asked her mother for assurance and reassurance that this had not happened. Sarah also worried that she might unintentionally lie and therefore avoided speaking and answering others' questions. Her OCD was thought to have originated when Sarah was around 4 years of age. Positively, her treatment led to significant reductions in Sarah's OCD: her CY-BOCS score dropped to '9' (mild) at the end of treatment and 5 (below clinical cut-off) at 6-month follow-up.

In terms of her developmental history, Sarah had good health in the neonatal period. She achieved her speech and language milestones at an appropriate age and was described as being competent in social interactions and enjoying imaginative play. Thus, an Autistic Spectrum Disorder was excluded. Sarah's parents reported that she initially found it difficult to separate from her mother at nursery school and that she continues to find school transitions difficult.

Regarding Sarah's family history, her father had a history of low mood and, additionally, describes himself as 'being particular about tidiness and cleanliness' and feeling compelled to 'check and recheck' his work. Sarah's mother describes herself as a 'worrier'. Neither of Sarah's parents, nor members of her wider family, had been diagnosed with a mental health problem.

V. FORMULATION

Based on a model proposed by Barlow [18], a longitudinal cognitive-behavioural formulation for BII phobia best described Sarah's experiences (see Figure 1).

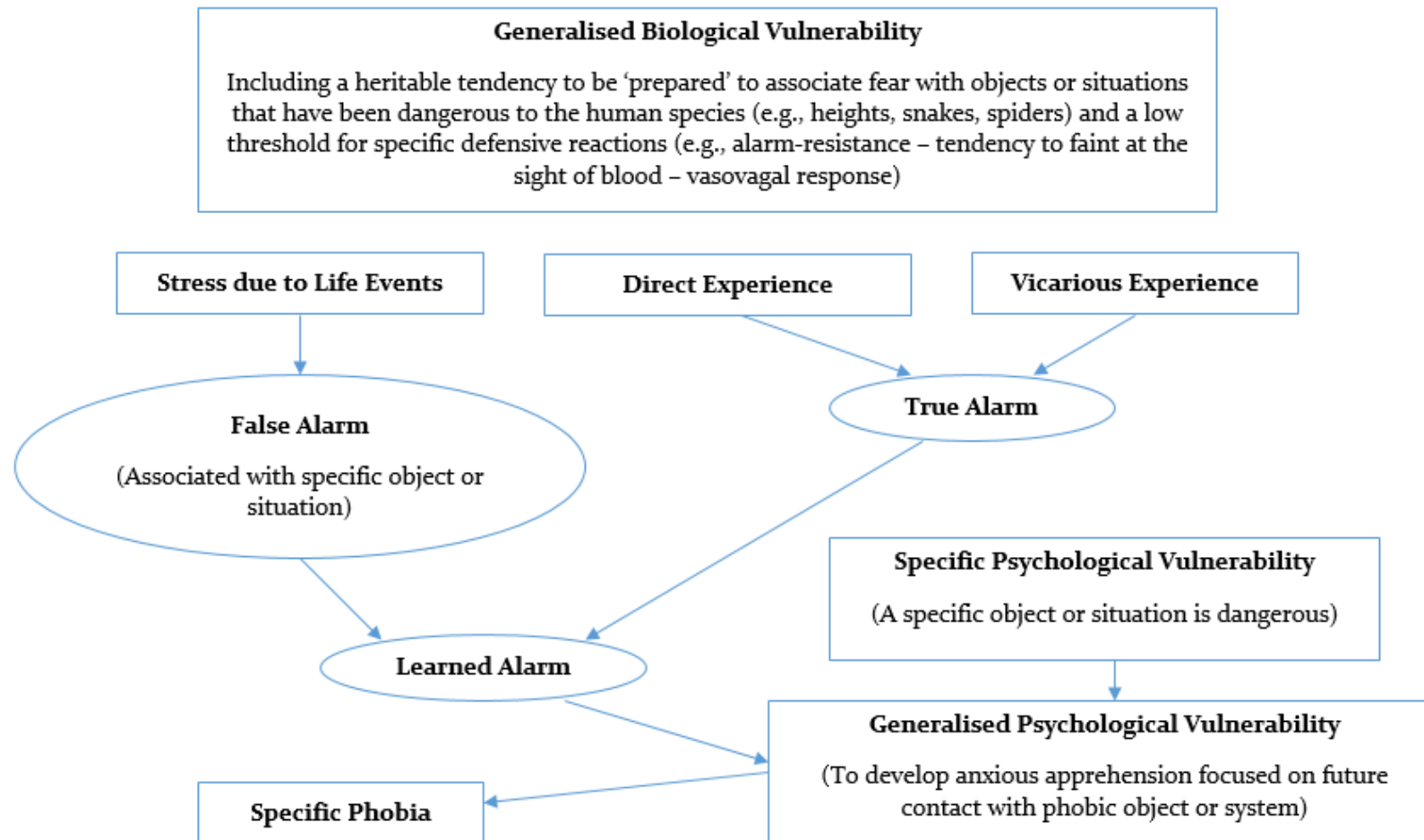


Figure 1: A model of the aetiology of specific phobia [18]

That is, a number of interacting vulnerabilities seemed to have facilitated Sarah's fear that she would 'feel floppy' (in her words) upon being presented with blood- and injection-related stimuli. Thus, a relatively non-specific generalised biological predisposition to experience anxiety and/or alarm responses, as well as familial transmission of anxiety, may have interacted with various distinct learning experiences, to cause her to associate fear and anxiety with blood, needles and associated stimuli.

Although it is clearly impossible to disentangle temperamental and environmental influences, Sarah's parents described her as having been a particularly anxious child, demonstrating fear and distress in relation to a range of stimuli (e.g., separation anxiety in relation to her mother, school transitions, OCD). Regarding familial transmission of anxiety, Sarah's having been exposed to an anxious mother, who herself described and modelled strong anxiety in the presence of blood- and injection-related stimuli, seems likely to have contributed to Sarah's interpretation of injections and blood-tests as things to be feared.

In terms of learning experiences, importantly, Sarah's one-month stay as an inpatient due to HUS in 2005 had, for obvious reasons, been an incredibly stressful episode in her and her family's life. During the course of this admission, Sarah underwent a number of successful blood-tests (i.e. that had not led to significant anxiety, fainting and avoidance), as well as peritoneal dialysis (during which a catheter is inserted into the abdomen). However, Sarah reported that she became increasingly fearful of having her blood taken over the course of this admission. It appears likely that Sarah's apparent generalised temperamental predisposition towards anxiety, together with the high level of anxiety she experienced during this episode, interacted such that Sarah came to associate a disproportionately high level of anxiety with blood and needles; indeed, this hypothesis was spontaneously endorsed by Sarah at assessment.

Although Sarah had successfully managed to undergo some vaccinations in the period between 2005 and 2012, these had been tolerated with progressively increasing anxiety and distress. It was during this period that she also began to faint (and demonstrate the signs and symptoms of possible imminent fainting) in response to blood-tests and injections. Sarah's possibly-inherited tendency to experience vasovagal syncope, in combination with these psychological vulnerabilities, likely led to her experiencing multiple 'false alarms' (substantial increases in anxiety) in the context of blood-tests

and injections and related stimuli/scenarios (e.g., the sight of needles and related medical equipment, GP surgeries and hospitals, having her blood-pressure taken, etc.).

Sarah's progressive false-alarm experiences culminated in a particularly anxiety-evoking incident, in Spring 2012, in which she had 'had a fit' (in her words) and fainted after an injection; Sarah reported feeling disorientated upon coming round. This incident proved to be so stressful that she refused/avoided any further blood-tests and injections from this point. Sarah's anxiety concerning blood- and needle-related stimuli had been exacerbated and maintained via negative reinforcement processes: thus, over a period of over 3 years (i.e. from Spring 2012 to Summer 2015), she experienced increasingly intense anxiety in the run-up to scheduled medical appointments (in which she was to have injections or her blood taken), in combination with increasingly powerful (short-term) feelings of relief associated with her subsequent avoidance of these appointments. Over time, Sarah's avoidance behaviours led to a generalised psychological vulnerability, that is, strong feelings of anxiety related not only to future exposures to blood- and injection stimuli in particular, but also to stimuli related to needles and injections (e.g., holding her arm out straight, in the manner preceding one having a needle inserted) and biological and medical stimuli more generally (e.g., a fear of fainting during Biology classes related to the circulatory system and of entering Hospitals and medically-related contexts, etc.).

VI. AIMS OF INTERVENTION

As stated above, Sarah had felt unable to undergo blood-tests, meaning that her Nephrology team could not assess her renal function. An appointment for an important and overdue blood-test had been scheduled for October 2015. It was therefore important that, by this time, Sarah's anxiety around blood and injections was reduced such that she was able to have her blood taken. It was also important for Sarah to undergo immunisations that she had missed. Additional aims were that Sarah would be able to tolerate watching television programmes/films containing blood, injections, injuries, etc., and that she would no longer feel the need to escape the classroom when certain topics (e.g., the circulatory system) were discussed.

VII. ASSESSMENT MEASURES

Sarah's intake assessment took place in December 2014. At this time, a number of measures of general wellbeing were administered to both her and her mother. The following measures were administered to Sarah: (i) the Affective Reactivity Index – Self-Report [ARI-S; 25]; (ii) the Mood and Feelings Questionnaire [MFQ; 26], and; (iii) the Strengths and Difficulties Questionnaire [SDQ; 27].

Sarah's mother completed: (i) the Affective Reactivity Index – Parent Version [ARI-P; 25]; (ii) the Depression Anxiety Stress Scales [DASS; 28], and; (iii) the SDQ – Parent-Report [29].

In addition, the team also rated her general level of functioning via the Children's Global Assessment Scale [CGAS; 30].

(For descriptions and scoring/interpretation of all the measures outlined above, see Appendix.)

The above measures are administered to all children and adolescents and their parent/s during intake assessment in the service. Her self- and mother-reported scores for each of these measures are presented in Tables 1 and 2, respectively.

Table 1: Sarah's self-reported outcome measures of general wellbeing at baseline

Measure	Score (classification, where relevant)
ARI-S:	
<i>Total score</i>	0
<i>Impairment score</i>	0
MFQ	2/66 (non-clinical range)
SDQ – Self-Report:	
<i>Emotional Problems Scale</i>	3/10 (non-clinical range)
<i>Conduct Problems Scale</i>	0/10 (non-clinical range)
<i>Hyperactivity/Inattention Scale</i>	3/10 (non-clinical range)
<i>Peer Relationship Problems Scale</i>	0/10 (non-clinical range)
<i>Prosocial Behaviour Scale</i>	8/10 (non-clinical range)
<i>Total Difficulties Scale</i>	6/40 (non-clinical range)

Note: ARI-S = Affective Reactivity Index – Self-Report; MFQ = Mood and Feelings Questionnaire; SDQ = Strengths and Difficulties Questionnaire.

Table 2: Sarah's parent-reported outcome measures of general wellbeing at baseline

Measure	Score (classification, where relevant)
ARI-P:	
<i>Total score</i>	0.17
<i>Impairment score</i>	0
DASS:	
<i>Depression</i>	0 (non-clinical range)
<i>Anxiety</i>	0 (non-clinical range)
<i>Stress</i>	3 (non-clinical range)
SDQ – Parent-Report:	
<i>Emotional Problems Scale</i>	3/10 (non-clinical range)
<i>Conduct Problems Scale</i>	0/10 (non-clinical range)
<i>Hyperactivity/Inattention Scale</i>	4/10 (non-clinical range)
<i>Peer Relationship Problems Scale</i>	2/10 (non-clinical range)
<i>Prosocial Behaviour Scale</i>	9/10 (non-clinical range)
<i>Total Difficulties Scale</i>	9/40 (non-clinical range)

Note: ARI-P = Affective Reactivity Index – Parent-Report; DASS = Depression Anxiety Stress Scales; SDQ = Strengths and Difficulties Questionnaire.

In addition to the more general measures administered, two measures specifically indexed difficulties related to Sarah's BII phobia.

Firstly, Sarah completed the self-report Fear Questionnaire [FQ; 31; see Appendix for a description of this instrument], which was used to index the severity of her BII. On this measure, Sarah scored as follows: (i) 11/40 for phobic avoidance, indicating that, overall, she was 'slightly avoidant' of BII-related situations; (ii) 7/40 for anxiety and depression, indicating that these feelings were somewhere between being 'hardly at all troubling' and 'slightly troublesome', and; (iii) 3/8 for global phobia, which lay mid-way between being 'slightly disturbing' and 'definitely disturbing'. Given that Sarah's BII phobia had led to her not only avoid important medical appointments, but was also impacting on her daily life in a variety of ways (e.g., leaving the classroom during Biology lessons, leaving the living room during television programmes and films), it is likely that her self-report somewhat underestimated the extent to which BII phobia was impacting on her life.

Secondly, Sarah self-rated her anxiety in relation to a number of BII-related scenarios, at several time-points across the intervention (see ‘Constructing a Hierarchy of Feared Stimuli/Situations’ in the ‘Intervention’ section, below).

Overall, therefore, Sarah’s BII phobia was a relatively specific concern for her, in the absence of more general and widespread emotional and psychological difficulties; thus, Sarah scored in the normal range, according to both herself and her mother, across the ARI-S, ARI-P, MFQ and SDQ. This was also reflected in her CGAS score of 63. Furthermore, her mother/family seemed not to have been adversely affected emotionally, as indicated by a DASS score in the normal range – although the family were somewhat involved in Sarah’s compulsions.

VIII. INTERVENTION

Sarah was offered 14 weekly hour-long sessions of CBT as an outpatient, in addition to three follow-up meetings post-treatment (at 1 month, 6 months and 12 months). The intervention was delivered jointly by both myself and a fellow Clinical Psychologist in Training. Sarah attended all 14 sessions on time, with her mother. She was pleasant, engaged and highly motivated in treatment throughout. Broadly, Sessions 1-2 were concerned with psychoeducation around anxiety and BII phobia; Session 3 involved introducing, discussing and practicing applied muscle tension (AMT) techniques; Session 4 was concerned with collaboratively constructing a hierarchy of feared BII-related stimuli/situations, and; Sessions 5-14 focused on Sarah being progressively exposed to each of these feared stimuli/situations in turn.

VIII.1) SESSIONS 1-2: PSYCHOEDUCATION

The psychoeducation in Sessions 1 and 2 included introducing and discussing the following: (i) the ‘fight or flight’ response and its evolutionary origins; (ii) the normative curve relating to elevations in anxiety (i.e. its rapid, initial increase from baseline to peak, followed by its more gradual reduction back to baseline); (iii) the (relatively high) prevalence-rates of phobias in general and of BII phobia specifically; (iv) the diathesis-stress model of BII phobia; (v) the cognitive/subjective, emotional, physiological and behavioural correlates of anxiety and avoidance in BII phobia and the relationship between these factors (i.e. a CBT-based cross-sectional formulation), and; (vi) the pre-syncopal and syncopal responses often observed upon exposure to feared stimuli in BII phobia, and its possible adaptive function (i.e. to prevent potential

toxins from travelling extensively around the body). The purpose of these discussions was to increase Sarah's understanding of and normalise her anxiety towards blood- and injection-related stimuli.

VIII.II) SESSION 3: AMT TECHNIQUES

Session 3 was given over to introducing and explaining the purpose of AMT techniques (i.e. to pre-emptively increase blood-pressure in anticipation of its dropping as part of a potential syncopal or presyncopal response on Sarah's exposure to BII-related stimuli), as well as demonstrating and practicing these with Sarah. In accordance with Mednick and Claar's [20] recommendations, and as per the method used by Chapman and DeLapp [3], Sarah was instructed to tense the muscles throughout her body for around 10-15 seconds, before then relaxing the muscles for around 20-30 seconds. This tensing and relaxing is alternated, so as to prevent either excessive arousal or excessive relaxation. Sarah was instructed to use AMT during the exposure tasks in Sessions 5-14.

VIII.III) SESSION 4: CONSTRUCTING A HIERARCHY OF FEARED STIMULI/SITUATIONS

In Session 4, in collaboration with Sarah, we developed a list of BII-related 'trigger' stimuli/situations that she found anxiety-provoking. For each one, we asked her to estimate the level of anxiety she experienced whilst in the presence of the stimulus/situation, on a scale from 0 (no anxiety whatsoever) to 10 (the greatest anxiety imaginable). On the basis of these discussions, Sarah described a number of stimuli as eliciting anxiety. These are shown, along with their associated anxiety ratings, in order from least (towards the top of the table) to most anxiety-eliciting (towards the bottom), in Table 3.

Table 3: List of blood-related trigger stimuli/situations and their associated anxiety ratings at baseline (ordered from lowest to highest, in terms of the degree to which they elicited anxiety)

Stimulus/situation	Anxiety rating (/10)
1. Sarah touching the inside of her own elbow* whilst talking about blood	4/5
2. Biology lessons (specifically, images and discussions of blood cells)	6
3. 'Knee'[†] myself	6
4. Personal, Sexual and Health Education (PSHE) involving blood and reproduction	7
5. Having a bad cut	7
6. Other people touching the inside of her elbow	7/8
7. 'Knee'[†] someone else	7/8
8. Having a blood-pressure cuff secured around her arm	7/8
9. Sarah's Mum cutting her own finger	8
10. Using a finger-pricker	8
11. Watching a television programme involving blood, wounds, injections, etc.	8
12. Watching a person having an injection	8/9
13. A video of someone having their blood taken	9
14. Visiting people in hospital	9
15. Having injections	10

* The cubital fossa.

† Sarah used the term 'knee' (herself or others) to refer to the act of touching the back of the knee. This had become a phobic stimulus due to her associating the back of the knee with the cubital fossa, due to their obvious similarities.

VIII.IV) SESSIONS 5-14: GRADED EXPOSURE TASKS

Sarah's hierarchy informed exposure tasks as part of a graded exposure hierarchy. The actual stimuli Sarah was exposed to in each session differed somewhat from those included as part of her hierarchy, on the basis of information that emerged as treatment progressed. In each week, we encouraged and facilitated Sarah's progressively exposing herself to increasingly anxiety-provoking BII-related stimuli in turn. The other Trainee and I modelled approach behaviours towards her phobic stimuli, prior to Sarah's engaging in these exposure tasks. As part of each task, Sarah rated her anxiety before, during and after; tasks were ended at the point at which her anxiety had reduced by at least half (in order to facilitate Sarah's learning that anxiety naturally reduces 'itself', that is, without her needing to engage in escape/avoidance behaviours). The other Trainee and I provided positive reinforcement whenever Sarah managed to complete a task. Sarah was given between-session homework tasks each week, which broadly involved her repeating the exposure tasks that we had completed in the session (as well as continuing to repeat exposure tasks from earlier weeks).

Given Sarah's 10-year history of BII phobia, there were, unsurprisingly, some challenging moments. For example, in Session 9 (which involved going into, exploring and discussing the instruments in a phlebotomy room), Sarah reported a significant increase in anxiety and pre-syncopal symptoms. This led her to suspend the expose and lean over, with her head on a stool to recover. However, after around five minutes, she reported feeling better, including her anxiety having reduced to 'o'. Positively, Sarah was able to return to the room and complete the task. This seemed to be a crucial milestone in therapy, in that (and according to her subsequent self-report) Sarah seemed to learn that she was able to tolerate and 'ride-out' high anxiety levels even in high anxiety-eliciting scenarios.

At the end of the course of treatment, the extent to which Sarah felt anxious at the prospect of exposure to (what had previously been) each of her trigger stimuli had significantly reduced (as can be seen in Table 6). She reported feeling '100% certain' that she would be able to undergo the scheduled blood-tests to assess her renal function.

IX. RESULTS

The outcome measures that Sarah and her mother were administered at baseline were administered again (that is, sent-out via post) at the end of her sessions, as well as at 3-, 6- and 12-month follow-up. Unfortunately, Sarah and her mother only completed these measures at the time of the 12-month follow-up. These data, together with their corresponding baseline scores, can be seen in Tables 4 and 5.

Table 4: Sarah's self-reported outcome measures at baseline and 12-month follow-up

Measure	Score (classification, where relevant)	
	Baseline	12-month follow-up
ARI-S:		
Total score	0	0
Impairment score	0	-†
EWSAS*:		
School/college studies and work	-	0/8
Daily skills	-	0/8
Social activities	-	0/8
Hobbies	-	0/8
Family and relationships	-	0/8
MFQ	2/66 (non-clinical range)	0/66 (non-clinical range)
SDQ:		
Emotional Problems Scale	3/10 (non-clinical range)	1/10 (non-clinical range)
Conduct Problems Scale	0/10 (non-clinical range)	0/10 (non-clinical range)
Hyperactivity/Inattention Scale	3/10 (non-clinical range)	1/10 (non-clinical range)
Peer Relationship Problems Scale	0/10 (non-clinical range)	0/10 (non-clinical range)
Prosocial Behaviour Scale	8/10 (non-clinical range)	10/10 (non-clinical range)
Total Difficulties Scale	6/40 (non-clinical range)	2/40 (non-clinical range)

Note: ARI-S = Affective Reactivity Index – Self-Report; EWSAS = Education, Work and Social Adjustment Scale [32; see Appendix for a description of this measure]; MFQ = Mood and Feelings Questionnaire; SDQ = Strengths and Difficulties Questionnaire.

* Not completed at baseline.

† Not completed at follow-up.

Table 5: Sarah's mother-reported outcome measures at baseline and 12- month follow-up

Measure	Score (classification)	
	Baseline	12-month follow-up
ARI-P:		
Total score	0.17	0
Impairment score	0	0
DASS:		
Depression	0/42 (non-clinical range)	1/42 (non-clinical range)
Anxiety	0/42 (non-clinical range)	2/42 (non-clinical range)
Stress	3/42 (non-clinical range)	1/42 (non-clinical range)
SDQ:		
Emotional Problems Scale	3/10 (non-clinical range)	0/10 (non-clinical range)
Conduct Problems Scale	0/10 (non-clinical range)	0/10 (non-clinical range)
Hyperactivity/Inattention Scale	4/10 (non-clinical range)	4/10 (non-clinical range)
Peer Relationship Problems Scale	2/10 (non-clinical range)	0/10 (non-clinical range)
Prosocial Behaviour Scale	9/10 (non-clinical range)	10 (non-clinical range)
Total Difficulties Scale	9/40 (non-clinical range)	4/40 (non-clinical range)

Note: ARI-P = Affective Reactivity Index – Parent-Report; DASS = Depression Anxiety Stress Scales; SDQ = Strengths and Difficulties Questionnaire.

As can be seen from Tables 4 and 5, although Sarah's general wellbeing measures were relatively low at baseline, at 12-month follow-up, there had nevertheless broadly been improvements in even these low scores. Furthermore, although Sarah had not

completed the EWSAS at baseline, her score at 12 months post-treatment indicated no impact of her difficulties upon her education and social adjustment.

In terms of clinician-rated measures, Sarah's CGAS score was rated as 63 at baseline and was 91 at 12-month follow-up – indicating that she was 'doing very well'. There was therefore a significant increase in her general functioning over the two time-points.

Table 6 shows Sarah's self-rated levels of anxiety in response to her feared situations at baseline, at the end of the sessions and at 12-months post-treatment.

Table 6: Anxiety ratings at baseline and post-treatment

Stimulus/situation	Anxiety rating (/10)		
	Baseline	End of Sessions	12-Month Follow-Up
1. Sarah touching the inside of her own elbow* whilst talking about blood	4-5	0	0
2. Biology lessons (specifically, images and discussions of blood cells)	6	0	0
3. ‘Knee’[†] myself	6	0	0
4. Personal, Sexual and Health Education (PSHE) involving blood and reproduction	7	2-3	2
5. Having a bad cut	7	0	0
6. Other people touching her arm	7-8	2	0
7. Knee[†] someone else	7-8	0	0
8. Having a blood-pressure cuff secured around her arm	7-8	0	0
9. Sarah’s Mum cutting her own finger	8	0	0
10. Using a finger-pricker	8	5	4
11. Watching a television programme involving blood, wounds, injections, etc.	8	0	0
12. Watching a person having an injection	8-9	0	0
13. A video of someone having their blood taken	9	5	4
14. Visiting people in hospital	9	0	0
15. Having injections	10	5	4

* The cubital fossa. † Sarah used the term ‘knee’ (herself or others) to refer to the act of touching the back of the knee. This had become a phobic stimulus due to her associating the back of the knee with the cubital fossa, due to their obvious similarities.

The table shows that Sarah's self-rated anxiety for each of her feared scenarios significantly reduced from baseline through to the end of the sessions. With a few exceptions, almost every scenario from numbers 1 through to 12 had reduced to zero by the end of sessions, regardless of its rating at baseline. Furthermore, these gains (or rather, reductions) had been maintained at 12-month follow-up. Those scenarios involving injections or needles more directly (e.g., numbers 10, 13 and 15) were, perhaps unsurprisingly, somewhat more resistant to change, showing smaller reductions. However, even the anxiety associated with these most-feared scenarios nevertheless had reduced by at least half by the end of the sessions, with these reductions being either maintained or reducing further at 12-month follow-up.

Sarah's self-reported phobic avoidance at baseline, and follow-ups, rated via the Fear Questionnaire, can be seen in Table 7.

Table 7: Sarah's self-reported phobic avoidance at baseline and the various follow-ups

Index	Baseline	Follow-Up 1	Follow-Up 2	Follow-Up 3
Phobic avoidance	11/40	3/40	1/40	0/40
Anxiety and depression	7/40	2/40	0/40	0/40
Global phobia rating	3/8	2/8	1.5/8	1/8

Consistent with the reductions in self-reported anxiety described above, these data show that Sarah's phobic avoidance of BII-related situations reduced, from being 'slightly avoidant' (a mean score of 2.2) at baseline to being 'not avoiding' (a mean score of 0) at 12-month follow-up. As stated earlier, it seemed that Sarah was underestimating her levels of avoidance and the global impact of her BII at baseline; the reduction from baseline to follow-up, and therefore her therapeutic progress, may 'in reality' have been somewhat larger. Similarly, Sarah also showed notable improvements in mood related to her BII phobia. Finally, she showed a substantial reduction in her global phobia rating, from being mid-way between 'slightly disturbing' and 'definitely disturbing' at

baseline, to being mid-way between 'phobia absent' and 'slightly disturbing' at 12-month follow-up.

Overall, therefore, Sarah's general wellbeing showed some improvement – though this had been relatively good at baseline. More importantly, there appeared to have been substantial improvements in her anxiety and phobic avoidance in relation to BII-related stimuli. Indeed, such was the reduction in her anxiety that, at 3-month follow-up, Sarah reported that she had managed to undergo her required blood-tests (i.e. to assess renal function). This had been her primary goal for her CBT treatment, and something that she had been building-up to for the previous five years. Crucially, although she had indeed been highly anxious (around 8/10), she had nevertheless managed to tolerate the experience. Sarah reported having used the AMT techniques and that, as a result, she had not felt faint. Sarah had also managed to have her blood-pressure taken, which had been an additional challenge in the past. Furthermore, and positively, the blood-tests indicated a full recovery and meant that she had consequently been discharged from the Renal Service.

These gains had been maintained at 6- and 12-month follow-ups. Specifically, Sarah had continued to regularly watch 'Casualty' (which she feels regularly exposes her to blood and injuries) and had ceased to avoid conversations or images involving blood-, injections- or related stimuli. Indeed, Sarah was able to describe some recent episodes in detail, a task that would have been incredibly difficult for her at the start of treatment. She had also been able to fully participate in Biology lessons – one of which had involved dissecting an eyeball. Although she reported still experiencing anxiety at the idea of having a blood-test of injection (5/10), she also stated that she would not avoid these. Finally, Sarah has even begun to consider Medicine as possible career (yes, honestly). As standard, Sarah's recent 12-month follow-up was her final appointment with the service, from which she has now been discharged.

X. DISCUSSION

The graded exposure programme delivered with Sarah was clearly successful. Together with the growing evidence-base suggesting the effectiveness of CBT via graded exposure (and using AMT) [e.g., 16, 17-19], it may be time for NICE to evaluate such evidence and to consider instituting guidelines recommending the use of such methods to address BII phobia.

Given the potential importance of high disgust-sensitivity in BII phobia [e.g., 12, 13-15], it may have been useful to include some measure of this [such as the Disgust Propensity and Sensitivity Scale-Revised; 33], in order to measure the impact of the intervention (or not) upon this construct (and vice-versa). The failure to include such a measure may be considered a limitation, and something that would be addressed were this single case design to be repeated.

A sometimes-challenging aspect of the intervention was the anxiety of Sarah's mother in relation to BII-related stimuli. Indeed, she often demonstrated clear signs of anxiety and refused to watch and take part in some of the exposures (e.g., observing mine and the fellow Trainee's blood being taken in the later sessions) as a result. This was interesting, as it provided obvious evidence in favour of Barlow's [18] diathesis-stress model of BII phobia. However, it was also challenging, as it of course set-up something of a conflict for Sarah, between the 'approach' demands of the graded exposure and the anxiety and 'avoidance' implicitly suggested by her mother. As it turned-out, Sarah's determination was such that she was not swayed by her mother's anxiety. Had this not been the case, it may have been helpful to explore Sarah's attending the sessions independently.

Nevertheless, that the intervention did not directly address Sarah's parents' anxieties may be considered a limitation. It is likely that this was an important factor, throughout the course of Sarah's childhood, in the development and maintenance of her BII phobia (as well as previous OCD diagnosis) [e.g., 18]. Sarah was 13 years old and will presumably be living at home with – and exposed to the anxieties of – her parents for at least a few years to come. That the present intervention did not address her parents' anxieties directly, therefore, may have a bearing on whether Sarah is able to maintain the gains made in treatment. That said, the present intervention's focusing rather specifically on Sarah's individual anxiety (to the exclusion of her familial context) is no

different to the (generally successful) approach(es) taken by Öst *et al.* [16], Craske *et al.* [19], Ayala *et al.* [17], amongst others. Indeed, this is a broader criticism that can be levelled at CBT approaches in general. However, it would be difficult for such a schedule of 14 weekly hour-long sessions to incorporate additional work on the difficulties of the index patient's significant other(s). Then again, it is hoped that Sarah's mother's apparent BII-related anxieties were addressed in some way, albeit indirectly, via her co-attending all of Sarah's sessions (e.g., by listening to the psychoeducation and observing the numerous exposure tasks).

XI. REFLECTIONS

Working with Sarah was sometimes challenging because fear of blood-tests/injections was something that I myself had struggled with previously – which made it relatively easy to empathise with her difficulties. Though I did not undergo any formal psychological therapy, I had managed to tolerate a number of regular blood-tests and immunisations as part of Occupational Health for a job. These had therefore served as a natural, *in vivo* exposure course and resulted in my becoming significantly less anxious about and more able to tolerate blood-tests and injections.

I believe that both my prior experience of BII phobia, as well as my having overcome this difficulty via a form of repeated exposures, influenced my practice throughout the intervention. In the initial exposure sessions, Sarah was struggling to fully engage with homework tasks (spec., putting-on and wearing plasters on her cubital fossae), and my empathy with her may have impacted on the extent to which I fully addressed and problem-solved these difficulties. I explored this in supervision and reflected on the crucial role of repeated exposures for alleviating BII phobia. From this, I was able to hold in mind the potentially limited impact of the intervention if Sarah was not more strongly encouraged to engage and persist with exposure tasks – especially at such times when her anxiety was clearly high. Overall, I feel that this contributed to my professional development, in terms of my ability to hold in mind treatment goals at times when the patient, as well as the therapist, may be finding aspects of an intervention challenging.

REFERENCES

1. Association, A.P., *Diagnostic and Statistical Manual of Mental Disorders: DSM-5 (5th Ed.)*. 2013, Washington, D.C.: American Psychiatric Association.
2. Bienvenu, O.J. and W.W. Eaton, *The epidemiology of blood-injection-injury phobia*. Psychol Med, 1998. **28**(5): p. 1129-36.
3. Chapman, L.K. and R.C. DeLapp, *Nine session treatment of a blood-injection-injury phobia with manualized cognitive behavioral therapy: An adult case example*. Clinical Case Studies, 2013: p. 1534650113509304.
4. Pitkin, M.R. and J.M. Malouff, *Self-arranged exposure for overcoming blood-injection-injury Phobia: a case study*. Health Psychol Behav Med, 2014. **2**(1): p. 665-669.
5. Marks, I., *Blood-injury phobia: a review*. Am J Psychiatry, 1988. **145**(10): p. 1207-13.
6. Ellinwood, E.H. and J.G. Hamilton, *Case report of a needle phobia*. J Fam Pract, 1991. **32**(4): p. 420-2.
7. Cox, D. and D.C. Mohr, *Managing difficulties with adherence to injectable medications due to blood, injection, and injury phobia and self-injection anxiety*. American Journal of Drug Delivery, 2003. **1**(3): p. 215-221.
8. Peinado Peinado, R., *[Is the prognostic significance of presyncope the same as for syncope?]*. Rev Esp Cardiol, 2004. **57**(7): p. 613-6.
9. Öst, L.G., U. Sterner, and I.L. Lindahl, *Physiological responses in blood phobics*. Behav Res Ther, 1984. **22**(2): p. 109-17.
10. Thyer, B.A., J. Himle, and G.C. Curtis, *Blood-injury-illness phobia: a review*. J Clin Psychol, 1985. **41**(4): p. 451-9.
11. Wood, S.R. and D.F. Tolin, *The relationship between disgust sensitivity and avoidant behavior: studies of clinical and nonclinical samples*. J Anxiety Disord, 2002. **16**(5): p. 543-59.

12. Wani, A.L., A. Ara, and S.A. Bhat, *Blood injury and injection phobia: the neglected one*. Behav Neurol, 2014. **2014**: p. 471340.
13. Sawchuk, C.N., et al., *Disgust sensitivity and contamination fears in spider and blood-injection-injury phobias*. Behav Res Ther, 2000. **38**(8): p. 753-62.
14. Olatunji, B.O., et al., *Disgust, anxiety and fainting symptoms associated with blood-injection-injury fears: a structural model*. J Anxiety Disord, 2006. **20**(1): p. 23-41.
15. Olatunji, B.O., et al., *Examination of the decline in fear and disgust during exposure to threat-relevant stimuli in blood-injection-injury phobia*. J Anxiety Disord, 2007. **21**(3): p. 445-55.
16. Öst, L.G., J. Fellenius, and U. Sterner, *Applied tension, exposure in vivo, and tension-only in the treatment of blood phobia*. Behav Res Ther, 1991. **29**(6): p. 561-74.
17. Ayala, E.S., A.E. Meuret, and T. Ritz, *Treatments for blood-injury-injection phobia: a critical review of current evidence*. J Psychiatr Res, 2009. **43**(15): p. 1235-42.
18. Barlow, D.H., *Anxiety and its disorders*. 2002.
19. Craske, M.G., M.M. Antony, and D.H. Barlow, *Mastering your fears and phobias*. 2006: Oxford University Press.
20. Mednick, L.M. and R.L. Claar, *Treatment of severe Blood-Injection-Injury Phobia with the applied-tension method two adolescent case examples*. Clinical Case Studies, 2012. **11**(1): p. 24-34.
21. Mataix-Cols, D., et al., *Symptom stability in adult obsessive-compulsive disorder: data from a naturalistic two-year follow-up study*. Am J Psychiatry, 2002. **159**(2): p. 263-8.
22. NICE. *Obsessive-compulsive disorder and body dysmorphic disorder: treatment (update)*. 2005; Available from: <https://www.nice.org.uk/guidance/cg31>.
23. Foa, E.B., *Cognitive behavioral therapy of obsessive-compulsive disorder*. Dialogues Clin Neurosci, 2010. **12**(2): p. 199-207.

24. Corrigan, J.J., Jr. and F.G. Boineau, *Hemolytic-uremic syndrome*. *Pediatr Rev*, 2001. **22**(11): p. 365-9.
25. Stringaris, A., et al., *The Affective Reactivity Index: a concise irritability scale for clinical and research settings*. *Journal of Child Psychology and Psychiatry*, 2012. **53**(11): p. 1109-1117.
26. Angold, A., et al., *Development of a short questionnaire for use in epidemiological studies of depression in children and adolescents*. *International journal of methods in psychiatric research*, 1995.
27. Goodman, R., H. Meltzer, and V. Bailey, *The Strengths and Difficulties Questionnaire: A pilot study on the validity of the self-report version*. *European child & adolescent psychiatry*, 1998. **7**(3): p. 125-130.
28. Lovibond, P.F. and S.H. Lovibond, *The structure of negative emotional states: Comparison of the Depression Anxiety Stress Scales (DASS) with the Beck Depression and Anxiety Inventories*. *Behaviour research and therapy*, 1995. **33**(3): p. 335-343.
29. Goodman, R., *The Strengths and Difficulties Questionnaire: a research note*. *Journal of child psychology and psychiatry*, 1997. **38**(5): p. 581-586.
30. Shaffer, D., et al., *A children's global assessment scale (CGAS)*. *Archives of General psychiatry*, 1983. **40**(11): p. 1228-1231.
31. Marks, I.M. and A.M. Mathews, *Brief standard self-rating for phobic patients*. *Behaviour research and therapy*, 1979. **17**(3): p. 263-267.
32. Lenhard, F., et al., *Internet-delivered cognitive behavior therapy for adolescents with obsessive-compulsive disorder: an open trial*. 2016(1932-6203 (Electronic)).
33. Van Overveld, W., et al., *Disgust propensity and disgust sensitivity: Separate constructs that are differentially related to specific fears*. *Personality and Individual Differences*, 2006. **41**(7): p. 1241-1252.
34. Cohen, N.J., N. Kolers, and S. Bradley, *Relation of global ratings of functioning with behaviour and development in delayed and disturbed preschoolers*. *Can J Psychiatry*, 1990. **35**(6): p. 514-8.

35. Mundt, J.C., et al., *The Work and Social Adjustment Scale: a simple measure of impairment in functioning*. Br J Psychiatry, 2002. **180**: p. 461-4.

APPENDIX: DESCRIPTIONS OF MEASURES COMPLETED AT BASELINE AND FOLLOW-UP

The Affective Reactivity Index [ARI; 25] – Self-Report and Parent Versions:

The concise, 7-item ARI scales index irritable mood. The individual items are scored 0 ('not true'), 1 ('somewhat true') or 2 ('certainly true'), with the first six items being summed to form the total score. The seventh item measures the extent of impairment owing to irritable mood, and is scored separately. The first six items are scored by dividing their sum by 6, by which one obtains a three-point scale corresponding to a three-level gradation of irritability severity (i.e. 'not true', 'somewhat true' or 'certainly true').

The Depression Anxiety Stress Scales [DASS; 28]:

The 42-item DASS simultaneously measures the negative psychoemotional states of depression, anxiety and stress in parents – each construct being assessed by 14 items. Respondents are asked to indicate the frequency with which they have experienced symptoms of depression, anxiety and stress during the previous week. Individuals respond to each item using a 4-point Likert scale, from 0 ('did not apply to me at all') to 3 ('applied to me very much, or most of the time'). Thus, potential scores for each subscale range from 0 to 42, with higher scores suggesting greater symptom-severity. Details of the scoring and interpretation of the DASS are given in Table A1.

Table A1: Scoring and interpreting the DASS

Range	Depression (D)	Anxiety (A)	Stress (S)
Normal	0-9	0-7	0-14
Mild	10-13	8-9	15-18
Moderate	14-20	10-14	19-25
Severe	21-27	15-19	26-33
Extremely Severe	<28	<20	>34

The Mood and Feelings Questionnaire [MFQ; 26] – Child Self-Report Long Version:

The 33-item Child Self-Report MFQ assesses current symptoms of depression. It consists of a series of descriptive phrases concerning how the respondent has been feeling and behaving within the past two weeks. The individual responds to each item by indicating 'not true' (scored '0'), 'sometimes' (1) or 'true' (2). A total score is determined by summing all items, with potential scores ranging from 0 to 66. A score of 25 or above is thought to optimally predict the presence of depressive symptoms.

The Strengths and Difficulties Questionnaire [SDQ; 27, 29] – Self- and Parent-Report Versions:

The SDQ is a 25-item questionnaire concerning the psychological attributes and behaviour of individuals aged between 4- and 17-years-old. The scale yields five subscales: 1) Emotional Problems (5 items); 2) Conduct Problems (5 items); 3) Hyperactivity/Inattention (5 items); 4) Peer Relationship Problems (5 items); and 5) Prosocial Behaviour (5 items). The scores from scales 1-4 can be added together to create a 'Total Difficulties' score. Details of the scoring and interpretation of the SDQ in 14-17-year-olds is given in Table A2.

Table A2: Scoring and interpreting the SDQ in 14-17-year-olds

	Normal	Borderline	Abnormal
<i>Parent-completed:</i>			
Emotional problems Scale	0-3	4	5-10
Conduct problems Scale	0-2	3	4-10
Hyperactivity/Inattention Scale	0-5	6	7-10
Peer Problems Scale	0-2	3	4-10
Prosocial Scale	6-10	5	0-4
Total Difficulties	0-13	14-16	17-40
<i>Self-completed:</i>			
Emotional Problems Scale	0-5	6	7-10
Conduct Problems Scale	0-3	4	5-10
Hyperactivity/Inattention Scale	0-5	6	7-10
Peer Problems Scale	0-3	4-5	6-10
Prosocial Scale	6-10	5	0-4
Total Difficulties	0-15	16-19	20-40

Children's Global Assessment Scale [CGAS; 30]:

The CGAS is a measure for assessing the severity of psychiatric disturbance and impairments in general functioning in children aged 4 to 16 years. It is scored from 1 to 100, with a score below 61 taken to indicate 'definite pathology' [34]. Summary decile descriptions are given in Table A3.

Table A3: Summary Decile Descriptions for the CGAS

Decile	Description
100-91	Doing very well
90-81	Doing well
80-71	Doing all right – minor impairment
70-61	Some problems – in one area only
60-51	Some noticeable problems – moderate impairment in several areas and unable to function in one area
50-41	Obvious problems – moderate impairment in most areas or severe impairment in one area
40-31	Serious problems – major impairment in several areas and unable to function in one area
30-21	Severe problems – unable to function in almost all situations
20-11	Very severely impaired – so impaired that considerable supervision is required for safety
10-1	Extremely impaired – so impaired that constant supervision is required for safety

The Fear Questionnaire [FQ; 31]:

The FQ yields scores for the extent to which the respondent is currently (i) avoiding phobic stimuli/scenarios; (ii) troubled by their phobia(s), and; (iii) globally disturbed by their phobia(s).

In the avoidance subscale, the respondent completes questions related to three forms of clinical phobia: agoraphobia, social phobia and blood and injury phobia. The respondent completes the questions relating to the phobia category/ies most relevant to them. Each category contains five factor-analytically-derived items, each rated on a scale of severity ranging from 0 to 8, where 0 denotes 'would not avoid it', 2 means

‘slightly avoid it’, 4 denotes ‘definitely avoid it’, 6 denotes ‘markedly avoid it’ and 8 means ‘always avoid it’. Scores are summed to give a total severity score ranging from 0 to 40, with higher scores indicating greater severity of avoidance.

Regarding (ii), the respondent completes questions relating to the extent to which they feel depressed, angry, tense, panicked and are having upsetting thoughts as a result of their phobia(s). Each item is rated on a 0 to 8 scale, where 0 indicates ‘hardly at all’, 2 means ‘slightly troublesome’, 4 denotes ‘definitely troublesome’, 6 means ‘markedly troublesome’ and 8 means ‘very severely troublesome’. Each item is scored individually.

In terms of (iii), the respondent rates the extent to which their phobia(s) is/are globally disturbing, on a single item, ranging from 0 to 8 (0 = ‘phobias absent’, 2 = ‘slightly disturbing/not really’, 4 = ‘definitely disturbing/disabling’, 6 = ‘markedly disturbing/disabling’ and 8 = ‘very severely disturbing/disabling’).

The Education, Work and Social Adjustment Scale – Child Version [EWSAS; 32]:

The EWSAS is an adaptation of the Work and Social Adjustment Scale [WSAS; 35] for children and adolescents, and measures the degree of impairment in five different areas of functioning (school, everyday situations, social activities, leisure time and family and relationships). The respondent rates the extent to which their difficulties affect their functioning in each domain, using a scale from 0 to 8 (0 = ‘my problem doesn’t affect this at all’, 2 = ‘my problem affects this slightly’, 4 = ‘my problem definitely affects this’, 6 = ‘my problem affects this a lot’ and 8 = ‘my problem affects this severely’).

CLINICAL CASE STUDY 4

'THAT'S WHEN THE SHARKS START CIRCLING'

COGNITIVE-BEHAVIOURAL FORMULATION AND THERAPY
FOR A COMPLEX ANGER PRESENTATION IN A 20-YEAR-
OLD MALE

SUPERVISED BY DR. NEIL HAMMOND

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I. LITERATURE REVIEW

I.I) AUTISM SPECTRUM DISORDERS

Autism Spectrum Disorders (ASD) are a range of neurodevelopmental conditions characterised by pervasive difficulties in social communication and interaction, together with restricted and repetitive patterns of behaviour, interests and activities [1]. Executive functioning deficits are also common [2], a recent study reporting those with ASD to be impaired across a number of indices measured [3]. The prevalence amongst adults has been estimated at 9.8 per 10,000, with males four times more likely to receive a diagnosis than females [4]. The aetiology of ASD is currently unknown. However, since genetic factors account for no more than 20-30 per cent of all cases, it is clear that complex genetic and environmental interactions are implicated in the remaining 70-80 per cent [5].

I.II) ATTENTION-DEFICIT/HYPERACTIVITY DISORDER

Attention-Deficit/Hyperactivity Disorder (ADHD) is a neurodevelopmental disorder characterised by pervasive and impairing symptoms of inattention and/or impulsivity and hyperactivity [1]¹. ADHD has a significantly detrimental impact across many aspects of behaviour, including on behaviour in the home and school/work performance [7]. ADHD has a relatively high prevalence-rate and is estimated to affect around 4 per cent of all children [8, 9]. It seems to persist, from childhood to adulthood, in around 50-60 percent of individuals [10-12]. Males are diagnosed more often than females, with the estimated male-to-female prevalence-ratio varying cross-culturally, from around 3:1 in Norway to 16:1 in Austria [13]. Although the aetiology of ADHD is under almost-continuous investigation, the evidence suggests an inter-play between multiple genetic and environmental risk factors [14].

An ADHD diagnosis confers vulnerability for a number of other comorbid DSM-V diagnoses [15]. Rates of comorbid depression have been estimated at around 28 percent, whilst between 10-37 per cent of individuals will go on to develop an alcohol and/or other substance use disorder [16, 17]. Approximately 20-50 per cent of children with

¹ Although the World Health Organisation (WHO) uses the term 'Hyperkinetic Disorder' (HD) in their ICD-10 (2015) to describe this set of behaviours, there is much overlap in terms of the operational criteria between ADHD and HD ([6]

ADHD go on to receive a diagnosis of conduct disorder during adolescence [15]. Percentages of comorbid personality disorder diagnoses in adults with ADHD range from 10-75 per cent, depending on sample characteristics [18], with anti-social personality disorder (ASPD) and borderline personality disorder (BPD) being observed most frequently [19, 20]. The mechanism(s) for the association between ADHD and personality disorders is/are at present unclear, although common genetic pathways [21], the impulsivity and hyperactivity seen in ADHD [22], as well as parenting styles [23] are all thought to be important predisposing factors [18].

ASD and ADHD commonly co-occur in individuals, with between 30 and 50 per cent of those diagnosed with the former exhibiting elevated symptoms of the latter [24, 25]. It is similarly estimated that features of ASD are observed in around two-thirds of individuals with ADHD [26].

1.III) ANGER MANAGEMENT ISSUES IN ASD AND ADHD

Anger management issues are relatively common in ASD [27], with rates of around 45 per cent having been reported [28]. In terms of potential mechanisms, those with ASD are known to be impaired with respect to inferring the mental states and emotions of those around them [29]. Combined with related and commonly-observed impairments in understanding non-verbal and reciprocal interactions, as well as ‘unwritten’ social rules, such difficulties can ‘set the stage’ for confusion and miscommunication. Furthermore, those with ASD are also impaired in terms of recognising and regulating their own emotions [i.e. alexithymia; 30] which, combined with executive functioning deficits, can lead to instantaneous physical responses, episodes of ‘blind rage’² and an ‘inability to recognise signals indicating that it would be appropriate to stop’ [6, p. 1204].

Individuals with ADHD experience greater levels of trait and state anger, relative to controls [32], and display this anger in more socially-inappropriate ways [33], such as ‘temper outbursts’ and emotional reactivity [34]. It stands to reason that elevated levels of anger and aggression in ADHD may be related to a central deficit in inhibitory control [35].

² ‘The rapidity and intensity of anger, often in response to a[n] [apparently] relatively trivial event, can be extreme’ ([31])

I.IV) ADDRESSING ANGER MANAGEMENT ISSUES IN ASD AND ADHD

The most recent National Institute for Health and Care Excellence (NICE) guidelines for addressing challenging behaviour in people with learning disabilities [NG11; 36] recommend a psychosocial intervention (provided that no coexisting mental or physical disorders, or problems relating to the physical or social environment, have been identified as triggering or maintaining the challenging behaviour). Most of the literature on anger interventions for those with ASD suggests using Cognitive-Behavioural Therapy (CBT). Sofronoff *et al.* [6] have outlined a modified CBT-based framework for addressing anger in children with ASD, involving the following components: (i) exploration of happiness and relaxation (i.e. ‘positive’ emotions); (ii) exploration of anger – specifically, its physiological, cognitive and behavioural correlates (and their inter-relation); (iii) common and personal ‘triggers’ for anger; (iv) introduction and development of an ‘anger thermometer’; (v) introduction and discussion of reality-/ probability-testing (e.g., alternative non-aggressive possible explanations for why people may behave in particular ways, etc.); (vi) discussion of strategies to alleviate feelings of anger (e.g., going for a run, listening to music, etc.), and; (vii) discussion of alternative, non-aggressive behaviours for expressing anger. Sofronoff *et al.*’s [6] programme also included ASD-appropriate modifications [cf. 31] (e.g., using visual materials and role-play to facilitate learning). In a Randomised Controlled Trial (RCT) using this protocol, Sofronoff *et al.* [6] reported significant reductions in parent-reported episodes of anger, as well as evidence indicating that strategies learned during treatment had become generalised to both home and school settings [see also 37]. Though such studies were conducted with children, their core components can be adapted for use with adults.

There are currently no specific NICE guidelines for addressing anger/aggression in ADHD. Given the strong links between ADHD and certain neurochemical abnormalities [38], drug treatment (methylphenidate) is recommended as the first-line intervention for adults with ADHD displaying either moderate or severe levels of impairment [CG72; 39].

II. THE PATIENT

The patient, 'Roshan' (not his real name), was a 20-year-old Asian-British male, of relatively large build, with a complex psychiatric presentation. He had diagnoses of ASD and ADHD. He was an only-child living with his mother. Although, at initial assessment, Roshan was undertaking a Business and Technology Education Council (BTEC) level 3 in IT, by the time he entered the ward, he had effectively discontinued his attendance. He and his mother owned a Staffordshire bull terrier, to which Roshan was highly attached.

III. THE PRESENTING PROBLEM

Roshan was referred to a tertiary inpatient service for ASD, due to high levels of anger and aggression. His anger was primarily directed towards – although not restricted to – family members.

At home, Roshan's anger manifested via destruction of property (e.g., destroying items of furniture/electrical equipment and causing structural damage) and verbal and physical aggression, the latter primarily towards his mother. Roshan described that he would 'throw [his mother] around, kick her and punch her'. In particularly violent episodes, he had even stamped and jumped on her after having knocked her to the ground. Such incidents had resulted in his mother having been hospitalised several times and needing ongoing physiotherapy. He had previously threatened his father with a knife.

His mother reported that Roshan had difficulties with daily functioning. The most common 'trigger' to his anger at home was being asked to complete necessary activities. Outside of the home, Roshan was also aggressive/violent towards peers when he perceived them as slighting him in some way (e.g., staring at him from across the street). At such times, he described his anger as overwhelming him, increasing 'from a 0 to a 10 immediately' and, 'turning [him] into an animal... I just want to inflict pain, hear people scream in agony and destroy everything.'

Roshan had no criminal convictions resulting from his violence. However, he had received a caution for 'assault occasioning actual bodily harm' (on his mother) in December 2014. Furthermore, he had been issued with 'Final Warnings' from two local Police forces for his domestic violence, meaning that any further offences may lead to

criminal prosecution without his mother having to press charges. At assessment, Roshan agreed that his aggressive outbursts were ‘unacceptable’, and consented to his inpatient referral in order for these issues to be addressed. Although numerous interventions had been previously undertaken, these had been of limited success, according to both Roshan and his parents.

IV. BACKGROUND

Roshan first came to the attention of Child and Adolescent Mental Health Services (CAMHS) in 2009, aged 12 years. He had had a number of exclusions from secondary school, due to ‘fighting, disrupting classes and persistently refusing to follow instructions.’ Roshan reported having previously been bullied at the school. He was also ‘struggling to adhere to boundaries’ at home (e.g., his aggression towards his parents). As a result, he was diagnosed with and underwent various treatments for ADHD, between 2009 and 2011.

In 2012, after treatments for Roshan’s ADHD had not resolved his anger/aggression, he was assessed for and diagnosed with an ASD. His parents first became concerned about his behaviour when he was 3 years old, describing him as having ‘a rigid need to be in control, inflexibility and extreme reactions to situations’. Roshan tended to play alone at Montessori school and, on one occasion, bit another child.

At around age 14/15, Roshan founded and developed a gang, along with a group of friends. Gang members behaved provocatively towards other gangs by, for example, entering their respective ‘territories’. Roshan stated that his gang affiliation made him feel ‘powerful’ and ‘unstoppable’. Subsequently – as his friends/other gang members came to be stabbed and even killed (when he was 16) – he came to realise the gang had become ‘really serious’. His school were instrumental in ending his gang affiliation at 16/17 years, encouraging Roshan and his friends to write and record music and thereby provide an alternative focus for their interests.

At assessment, Roshan was assessed for the potential presence of a range of mood and anxiety disorders and schizophrenia, but these were excluded. He was not taking any psychotropic medication, was a non-smoker and was neither abusing alcohol nor taking/abusing any other (i.e. illicit) drugs.

V. FORMULATION

On the basis of Roshan's presenting difficulties and background information, collected at assessment, and initial discussions with him from our early sessions, his case was formulated longitudinally as follows (see Figure 1).

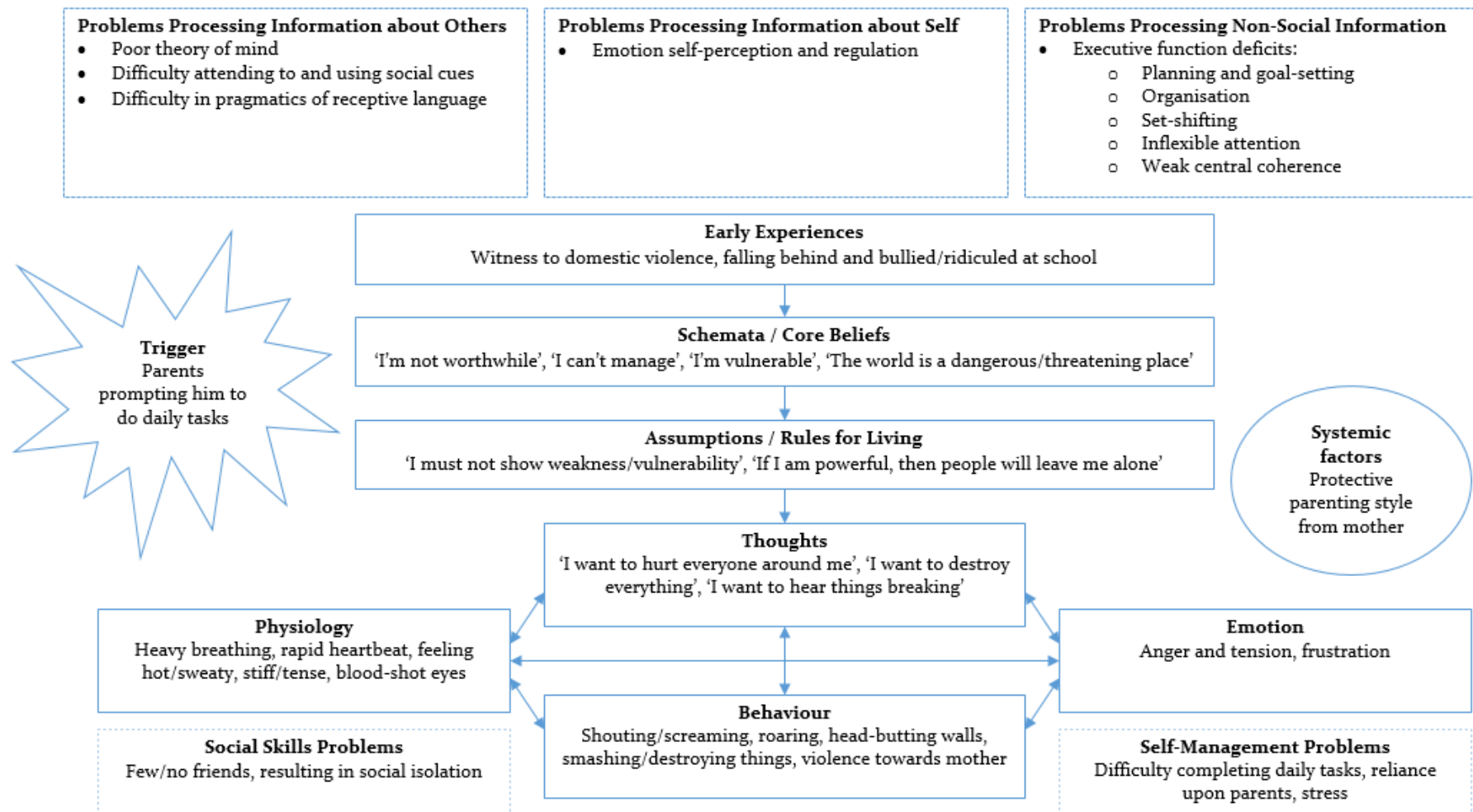


Figure 1: Longitudinal formulation of Roshan's anger and aggression presentation

Roshan seemed to demonstrate a number of neurological difficulties typical of ASD – that is, in the processing of information relating to both others and oneself, as well as executive functioning deficits.

In terms of Theory of Mind ability (or ToM), Roshan often becomes angry when in the car and another driver toots their horn. He tends to assume that the driver is tooting at *them*, and ‘is unable to see that they may be tooting someone else or making a general protest.’ Also, Roshan stated that, if his mother shouts/screams (to stop) whilst he is attacking her, this ‘pisses [him] off even more and makes [him] hit her even harder.’ Thus, he seemed not to recognise that she may be shouting due to fear and/or pain, and might have been trying to bring the violence to a close, rather than – as he believed – deliberately trying to provoke him further.

Roshan also seemed to have enduring difficulties understanding social information, including appropriate behaviour in a given situation. He had bitten another child at primary school. Roshan often talked at length about tangential/irrelevant topics despite a number of verbal and non-verbal prompts, and had similar difficulties recognising cues indicating the end of the session. His mother reported that Roshan often misunderstands what others say to him (e.g., failing to understand jokes and interpreting things in a literal manner).

Roshan had clear difficulties in emotion self-perception and regulation – seemingly an enduring risk-factor for his aggression. Thus, he described not being aware of when his anger was building-up, experiencing these episodes as having a rapid onset (making his anger ‘go from a 0 to a 10 immediately’). He also seemed to have significant difficulty in perceiving his emotions more generally, frequently describing himself as being ‘only angry or neutral’, and denying experiencing other emotions.

There was also evidence of executive functioning deficits. His attention was reported as being poor by both himself and his mother. He also seemed to struggle with impulsivity. Roshan reported having had difficulty with exams at school, due to responding too quickly to them, not thinking through what the questions were asking. During our sessions, I noticed that he would start to respond to a question immediately after my having asked it (speaking rapidly, with little ‘space’ between sentences and ideas), appearing not to have fully processed what I had asked. He also found planning and organisation very difficult, to the extent that his father took on much/all of the

responsibility for Roshan's schedule. Similarly, he would frequently misplace/forget items (e.g., his keys, phone/laptop chargers).

It seems likely that, due to a number of relatively difficult early experiences, Roshan's self-concept was relatively poor. Firstly, he had struggled with educational attainment from primary school onwards. Combined with his difficulties in ToM and social interactions (his mother described him as having been a very quiet child and not very sociable), it is perhaps unsurprising that he became isolated from his peers and experienced significant episodes of bullying during primary and secondary school.

In addition, Roshan's parents' relationship was reportedly disharmonious, Roshan being witness to physical violence between his parents from a young age. In 2007, Roshan's parents separated 'acrimoniously' and subsequently went on to live in separate parts of the country. Roshan's living arrangements had since been unstable, being successively transferred between his parents at various stages. From around 2013 (i.e. including the time of the assessment and intervention), Roshan had been living with his mother.

A number of negative core beliefs were hypothesised. His difficulties in school attainment and in managing everyday tasks could have fostered a belief of his not being capable/good enough. He told me, on a number of occasions, 'I haven't got the concentration or the motivation to sit down and study for exams, so what's the point?' Roshan's experience of being bullied may have led to his developing beliefs such as his being vulnerable, unacceptable to others and/or that other people cannot be trusted. It seems likely that being successively transferred between his parents may have served to reinforce such possible beliefs. His (regular) exposure to domestic violence, together with his time as part of a gang, may have led to a perception of the world as dangerous/threatening, and anger and aggression as adaptive responses within this.

In terms of Roshan's assumptions/rules for living, he seemed to think it important not to appear weak or vulnerable as, in his words, 'that's when the sharks start circling'. Such thoughts may have precipitated his gang membership. It seems likely that the rather concrete nature of this 'solution' to possible feelings of vulnerability may have been related to his ASD. It is also reasonable to conclude that his witnessing domestic violence may have led to his viewing aggression as a valid means of expressing oneself and resolving perceived 'threats' (the term 'threats' is used here in the broadest sense,

including both perceived overt challenges/ridicule from others, through to perceived implicit criticisms associated with requests from others (e.g., to clean his room, complete schoolwork, etc.)).

Roshan's parents reported that their prompting him to engage in typical daily-living tasks often served as triggers for his anger. Such prompts would generally remind Roshan that he has not carried out a given task, possibly activating hypothesised core beliefs around being unable to manage. His self-management problems could have served as further 'evidence' for such beliefs. More systemically, his mother described her parenting style as having been/being protective ('I never let Roshan out alone'). Although an understandable response to Roshan's vulnerabilities, such behaviour may have contributed to and reinforced his hypothesised perceived low self-efficacy. Perceived slights from peers may have triggered his anger due to sensitivity towards potential signs of threat, related to a perception of the world as dangerous.

Roshan was able to describe a number of symptoms of anger in response to triggering stimuli. At such times, Roshan reported feeling suddenly overwhelmed by his anger ('going from a "o" to a "io" instantly') and that he would 'become out of control... like a wild animal'. The episodes typically lasted around 30-40 minutes, after which Roshan would feel exhausted and fall asleep. Functionally, behaving thus may have served to make Roshan feel temporarily powerful, as well, perhaps, as serving as an avoidance from difficult thoughts and feelings around vulnerability.

In summary, Roshan's neurological difficulties and challenging early experiences may have interacted such that his pervading view of the world was of a threatening and dangerous place within which one is at an ongoing risk of 'attack' (including perceived criticism) from others. It appears that his rigid/concrete thinking style, together with his having witnessed significant episodes of domestic violence, may have led him to adopt aggressive behaviours in anticipation of and in response to such perceived attacks.

VI. AIMS OF INTERVENTION

The overall aim of the intervention was to reduce Roshan's aggressive outbursts (and the attendant risks posed to others). More specifically, the aims were to allow Roshan to: (i) become more aware of 'triggers' for his anger; (ii) become more aware of signs and symptoms that his anger is increasing (before the point at which it went to a '10' and he 'turn[ed] into an animal'); (iii) learn strategies for diffusing/reducing his anger, and; (iv) learn alternative behaviours for managing anger-inducing situations (e.g., to communicate dissatisfaction without aggression or violence).

VII. INTERVENTION

A modified form of CBT, incorporating the core elements described by Sofronoff *et al.* [6] but tailored to Roshan's presentation and formulation and using ASD-appropriate adaptations (e.g., the use of metaphors to aid understanding, using a whiteboard to summarise ideas/draw-out diagrams and reduce the 'talkiness' of sessions), was delivered. The intervention took place over 8 weekly, hour-long sessions, with 'homework' tasks between sessions.

In the first half of the sessions, we explored the following. Given Roshan's difficulties in noticing the 'warning signs' of increasing anger, our sessions included a specific focus on common and personal triggers to anger and its cognitive and physiological correlates, as well as the relationships between these factors (i.e. a cross-sectional formulation). In order to develop Roshan's understanding of how anger can increase, we used the metaphor of a 'pressure-cooker' to describe how anger can (continue to) build-up and, if the 'pressure' is not released (e.g., via various tension-reduction strategies), cause the lid to 'blow-off' (e.g., smashing and destroying property, verbal and physical violence aimed at his mother). As part of this, we explored the 'fight or flight' system and used the 'broken car alarm' metaphor to explain how one's threshold for the triggering of anger may become lowered (in Roshan's case, due to e.g., his experiences of gang violence). We also thought about how anger is a natural part of the human experience, its evolutionary origins, as well as its advantages (e.g., to signal discontent with others and thereby facilitate goal-directed behavior to address the source of discontent) and disadvantages (e.g., injuring others, police involvement). Throughout sessions, I looked for potential 'openings' to discuss Roshan's potential underlying thoughts and feelings associated with anger.

We spent time thinking about factors that can commonly and personally influence anger. We explored how symptoms of ASD can sometimes make anger more likely (e.g., how ToM difficulties can sometimes lead people to interpret situations as threatening). Also, we thought about the impact that low mood can have on anger, and the importance of engaging in tasks and activities known to improve mood (e.g., sleep hygiene, engaging in regular exercise and eating a healthy, balanced diet).

In the latter half of the sessions, we thought about strategies to reduce anger and aggressive behavior. First, we explored common and potential personal strategies for reducing angry feelings. Roshan found being on his own in his room, listening to (reggae) music and going for a walk with his dog particularly helpful strategies for calming himself down. Following this, we thought what we can do at those times when it might not be possible/practical to reduce internal angry feelings via the above strategies. We talked about how we can respond to internal anger in three broad ways: passively (i.e. 'putting-up' with something that we are unhappy with), aggressively or assertively (i.e. making it clear that we are unhappy with something in a clear, non-aggressive way). In order to illustrate these forms of responding, [an Assistant Psychologist] and I performed role-plays to demonstrate each form of behavior and asked Roshan to identify each, followed by a discussion of their respective merits and drawbacks.

VIII. MEASURES

The following measures were administered to each party at baseline (before the intervention had begun) and again at the end of the intervention.

Roshan completed the Novaco Anger Inventory – Short Form [NAI-25; 40] which measures the degree of anger felt in a range of anger-inducing situations. Each question is rated on a 5-point Likert scale, from 'very little' (0) to 'very much' (4). A total score is calculated by summing all items. A Cronbach's alpha of 0.96 has been reported for the scale [40]. Items describing scenarios that Roshan rated as eliciting high levels of anger were explored as part of the intervention (e.g., 'you are in a discussion with someone who persists in arguing about a topic they know very little about'). Roshan scored 29/100 at baseline.

Roshan also completed the Anger-Out (MAO) subscale and Anger-In (MAI) subscales from the Multidimensional Anger Inventory [MAI; 41, 42]. These subscales respectively

measure the tendency to express and suppress anger. Each item is rated on a 5-point scale, ranging from 'completely undescriptive of me' (1) to 'completely descriptive of me' (5). Adequate internal consistency for these subscales has been reported [41, 43]. Roshan completed the MAO and MAI at baseline and the end of the intervention. At baseline, he scored 8/10 for the MAO subscale [that is, 0.91 standard deviations, or SDs, above the mean, M, based on norms published by 44] and 12/25 on the MAI (0.05 SDs above the mean).

Roshan's mother also rated his anger and aggression via other-reported versions of the NAI-25 and MAO and MAI. At baseline, Roshan's mother scored him as 64/100 on the NAI-25, 9/10 (1.42 SDs above the M) for the MAO and 18/25 (1.52 SDs above the M). Thus, although there was some consistency between Roshan's and his mother's ratings for the MAO, his mother's ratings were somewhat higher for the NAI-25 and the MAI.

In addition, Roshan's primary nurse on the ward was asked to rate him on the other-reported version of the MAO subscale after having had him on her caseload for around a month (she was not asked to complete the NAI-25 or MAI at baseline as these scales depend on a good knowledge of the person being rated, which it was felt she could not have at that point). His primary nurse scored Roshan 2/10 (that is, 2.10 SDs below the M). Interestingly, this was substantially lower than both Roshan's and his mother's score.

VIII.1) NEUROPSYCHOLOGICAL ASSESSMENT

In addition, and as part of an extended assessment (conducted across a number of meetings running parallel to the intervention sessions), a battery of neuropsychological tests were administered to explore various aspects of Roshan's cognition.

Roshan was firstly administered the Wechsler Adult Intelligence Scale – UK Fourth Edition [WAIS-IVUK; 45] in order to measure his general intellectual functioning. After that, he completed the following tests to index the range of executive functioning abilities: (i) the Trail-Making Test [46], which measures attention and processing-speed; (ii) the Delis-Kaplan Executive Functioning System [D-KEFS; 47] Letter Fluency, Category Fluency and Category Switching subtests, to assess verbal fluency and set-switching; (iii) the Hayling Sentence-Completion Test [48], to index initiation speed

and inhibitory control, and; (iv) the Brixton Spatial Anticipation Test [48], a measure of cognitive flexibility and the ability to discern patterns.

IX. DIFFICULTIES

Although Roshan engaged well throughout the first half of the sessions, some difficulties began to emerge after around the half-way point. Specifically, he would often rapidly digress on to other, somewhat tangential subjects (e.g., Chelsea Football Club [and football in general], the pragmatics and difficulties of looking after his Staffordshire Bull Terrier and, most frequently, his strong dislike of his mother and difficult relationship with her). Once Roshan had ‘got started’ on a digression, it was incredibly difficult to ‘pull him back’ from it. His ASD notwithstanding, Roshan often seemed to understand that I was trying to refocus the conversation and seemed to actively resist my doing so. Thus, he would often speak even more quickly and adhere to his digression more forcefully, resulting in the sessions often feeling to me like something of a tug-of-war. I discussed this in supervision and problem-solved various strategies to mitigate these interruptions (e.g., Roshan and I agreed that, if I felt that we were going ‘off-topic’, I could interrupt him and redirect him to the agenda). However, such attempts had little impact, reducing the amount of therapeutic time per session.

Although Roshan had come into therapy saying that his anger/aggression was ‘unacceptable’, he subsequently seemed ambivalent about whether/how much he agreed that he had difficulties with anger and also whether/how much he wanted to change. He often remarked that, if one chooses not to respond to provocation with (high levels of) aggression, ‘that’s when the sharks start circling’, leaving one open to further ‘attacks’ (both from the original provocateur, as well as third-party ‘onlookers’). Whilst we were exploring the disadvantages of aggression and violence, and the conversation had turned to potentially being arrested, going to jail, etc., Roshan talked at length about strategies for dealing with police questioning and specifically, how not to implicate oneself (e.g., ‘You just have to make sure you don’t say anything – you just say “No comment”, “No comment” to everything they ask...’). Such comments were manifestations of a general and consistent ‘gangster’ persona that Roshan had adhered to and did not seem to wish to part with. Roshan’s history of witnessing domestic violence and his having been involved in a gang (and its associated violence) may have

made it difficult for him to contemplate/accept potential alternative behaviours to aggression.

Related to the above, but perhaps most importantly, as the sessions progressed, it became increasingly clear that Roshan seemed to have an intense dislike, possibly even hatred, towards his mother. Both Roshan and his mother described his feeling no guilt or remorse about his quite brutal levels of violence towards her (e.g., dragging her by the hair across the floor, throwing her into wine-racks, jumping on her neck whilst she lay on the floor). Indeed, such were his apparent levels of contempt and antipathy towards his mother that, when discussing these incidents, rather than remorse, he would often seem to become re-irritated and re-angry at his recollection of the thing(s) she had said or done to provoke him on a given occasion. Throughout the sessions, he repeatedly stated that he has ‘no bond with her’ and that, ‘she is just like any other person on the street to me.’ When asked to reflect on good things about her, Roshan said that ‘there isn’t anything.’ Indeed, by far the majority of his digressions concerned his mother. Roshan described her as obstinate, stupid, confrontational and provocative, and felt that she did things deliberately to irritate and anger him. Furthermore, from discussions with Roshan and his mother, it became increasingly clear that Roshan’s anger and aggression appeared to occur almost exclusively in the context of the relationship with his mother. He often said that ‘*she* [his mother] should be in here [the ward] having this [the sessions], instead of me.’

Roshan also strongly disagreed with and rejected his diagnosis of ASD and, as an informal patient, decided to spend increasingly less time on the ward. After a relatively short time, he was only coming to the ward for his anger sessions (e.g., he refused to remain on the ward on days when he did not have an anger session [which was most days, as he had one session per week] and did not attend any other ward groups, such as Social Skills Training and Understanding Your Emotions groups).

X. RESULTS

Roshan’s self- and other-reported outcome measure scores at baseline and at the end of the intervention are shown in Table 1.

Table 1: Roshan's self- and other-reported outcome measure scores at baseline and at the end of the intervention

Measure	Baseline	End of intervention
<i>Self-reported:</i>		
<i>NAI-25</i>	29/100	13/100
<i>MAO</i>	8/10	5/10
<i>MAI</i>	12/25	9/25
<i>Mother-reported:</i>		
<i>NAI-25</i>	64/100	Not collected
<i>MAO</i>	9/10	7/10
<i>MAI</i>	18/25	15/25
<i>Primary nurse-reported:</i>		
<i>MAO</i>	2/10	Not collected

Note: NAI-25 = the Novaco Anger Inventory – Short Form; MAO = Multidimensional Anger Inventory Anger-Out subscale; MAI = Multidimensional Anger Inventory Anger-In subscale.

As can be seen from the table, at the end of the sessions, Roshan rated himself around 55% lower (relative to baseline) for the degree of anger that he believed various anger-inducing scenarios (would) elicit in him (the NAI-25). He rated himself around 38% lower for the extent of his aggressive behaviour, and 25% lower for the amount of anger he generally felt. Throughout the intervention, Roshan showed clear evidence of learning from the sessions (e.g. by regularly making reference to subjects that we had explored together), both in our sessions and in his discussions with another ward Psychologist. At the end of the sessions, Roshan said that his anger and aggression had ‘clearly got less... I mean, it’s obvious, you can even ask my Mum.’

Roshan’s mother rated him around 22% lower for aggression (MAO) and reported a perceived 17% reduction in his levels of anger (MAI). His mother verbally completed the MAO and MAI during a phone-call at the end of the intervention. Due to time-constraints, it was not possible to collect his mother-reported NAI-25 during this same phone-call. Unfortunately, despite a number of subsequent attempts, she could not be contacted thereafter to provide this. Thus, though Roshan’s mother reported a reduction in his anger and aggression, this was not as pronounced as Roshan’s self-ratings. Nevertheless, she also spontaneously verbally reported that Roshan’s aggression had reduced.

Roshan's primary nurse felt that, due to his spending so little time on the ward, she did not have enough information to complete the MAO again at the end of the intervention. That said, although, in any given week, Roshan spent little time on the ward, he did, in total (i.e. total number of hours from intake through to discharge) spend a considerable amount of time there (and occasionally participated in ward sports groups and group lunches). Throughout this time, he was not involved in any angry or aggressive incidents.

Overall, therefore, despite the difficulties described above, the intervention seemed to have had an effect on Roshan's anger and aggression. Nevertheless, there were two occasions during the intervention on which Roshan had physically attacked his mother (once following his mother's locking his dog outside the front door and its having wandered-off, and once following a sudden change of plans). Though it may sound facile, both Roshan and his mother (independently) stated that his aggression on these occasions had 'not been as bad as before' (i.e. it had consisted of grabbing and shoving her, rather than throwing her around and jumping onto her).

X.1) NEUROPSYCHOLOGICAL ASSESSMENT RESULTS

On the WAIS-IV^{UK}, Roshan's verbal comprehension, working memory and processing speed performances all fell into the Average range, whilst his perceptual reasoning performance fell into the Low Average range. He achieved an overall IQ score of 96 (Average range). Although Roshan's Verbal Comprehension was somewhat greater than his Perceptual Reasoning, his performance revealed no notable areas of particular strength or weakness. (For a detailed assessment report, see Appendix 1.)

Performance across the range of executive functioning tasks was satisfactory. On both Parts A and B of the Trail-Making Test, Roshan performed within the Average range. On the D-KEFS Letter Fluency subtest (fluency for words beginning with 'F', 'A' and 'S'), he performed within the High Average range. On the Category Fluency and Category Switching subtests, he performed within the Average range. On the Hayling Sentence-Completion Test, Roshan scored within the Average range. On the Brixton Spatial Anticipation Test, he also scored within the Average range. (For all raw scores, see Appendix 2.) These results therefore did not indicate the presence of *any* deficits in any area of executive functioning (i.e. in attention and/or processing speed, verbal fluency, set-shifting, inhibitory control or cognitive flexibility).

Overall, surprisingly and despite his ADHD diagnosis, there were no indications of any focal neuropsychological impairment. Importantly, his performance on executive tests suggested that he may no longer meet criteria for a diagnosis of ADHD. This is consistent with research indicating that around 40-50 percent of those diagnosed with ADHD in childhood do not meet criteria in adulthood [10-12].

XI. DISCUSSION

The apparent relative success of the CBT intervention aside, by the end of the sessions, it seemed that relational/systemic factors were playing a greater part in his presentation than had seemed the case at the beginning. Although at assessment Roshan's anger had seemed relatively pervasive, it gradually became clear that his anger occurred primarily within the context of the relationship with his mother. Not only did Roshan report an intense dislike of her, he also viewed other members of the team with whom she had communicated as having been manipulated and 'poisoned' by her (that is, poisoned *against him*). Thus, due to his mother having attended ward-round a number of times, he refused to attend any weekly ward-rounds. On the contrary, Roshan did not seem to view me as having been so poisoned, due to my having worked primarily with him – indeed, he regularly stated that I was one of the few people 'who understood'. This seemed to indicate both that Roshan did not feel that, in general, his perspective had been taken into account (by mental health professionals), and that his mother's viewpoint had been given prominence. This impression may have been reinforced by the CBT intervention, with its key assumption that factors intrinsic to the individual are 'problematic' and thus targets for change. Given that the results of the neuropsychological assessment indicated normal functioning in attention and other executive functions (and an absence of adult ADHD), it seemed that his persistent excessive talking and disruptions of sessions may have been functional. The function of this behaviour was interpreted as a means via which Roshan was partially rejecting the intervention, due to disagreeing that his anger problems were simply 'located within' him. Also, Roshan may have viewed the sessions as an opportunity to not only make it clear to *me* that his mother was a significant part of his anger issues, but also that I may then communicate this to other team members and thus act as a representative for him in the ward rounds that he refused to attend.

Roshan's behaviour in sessions, and the impact that this had upon me, also seemed to provide important information for the possible dynamics of the apparently conflict-

dominated interactions with his mother. His excessive talking often left me feeling frustrated, and this may be the effect that he has upon his mother. Notably, however, several team members (including myself) who had interacted with Roshan's mother observed that it seemed relatively difficult to have a reciprocal exchange with her, and that she, too, seemed not to pick-up on non-verbal cues and to be somewhat dominant in conversation. A number of interpretations for this are possible, which are not elaborated upon here. Regardless, overall, the evidence seemed to indicate that, in addition to various factors intrinsic to Roshan (e.g., his ASD), the dynamics between Roshan and his mother were a significant trigger and maintenance factor in Roshan's anger – and therefore needed to be specifically addressed.

In addition, and complicating the picture further, it seemed that Roshan may have been traumatised from having witnessed significant episodes of domestic violence throughout his childhood and having witnessed and been involved in gang violence. Towards the end of my sessions with Roshan, his mother informed me that, when he was around 9 years of age, Roshan witnessed his father attack his mother, hitting her over the head with a 'heavy lampstand' in their bedroom and causing her to bleed profusely, over the bedsheets and carpet. According to her, Roshan had stood on the threshold watching this, and had remained 'rooted to the spot, staring' for around 15 minutes following the incident. During his time in a gang, one of Roshan's friends had been stabbed and killed. Viewed against the wider, threat-saturated context of his life history, Roshan's (somewhat rigid) adherence to a gangster persona, his difficulties in contemplating alternatives to aggressive behaviour and his preoccupation with/vigilance for signs that 'the sharks' are circling him, seem understandable. Although I allowed time during the sessions to explore these events and the impact that they may have had on Roshan, he did not seem ready to discuss this.

As stated above, Roshan and his mother both reported that he experienced little or no remorse for the aggression and violence that he directed towards his mother. As described in the literature review, individuals diagnosed with ADHD in childhood often go on to develop anti-social personality disorder (ASPD) or borderline personality disorder (BPD) in adulthood – although the precise nature of the relationship between ADHD and personality disorders seems unclear at present [18]. Nevertheless, it is possible that Roshan's apparent lack of guilt may have reflected the presence of possible ASPD, given that this is a key criterion in diagnosis of the disorder [49]. That the presence of ASPD or another personality disorder was not formally

assessed may be considered a limitation, and would be addressed were this single case design to be repeated.

Overall, the effectiveness of the CBT intervention reported here may have been somewhat limited, due to the influence of the factors outlined above. However, it nevertheless allowed Roshan to explore some important aspects of his anger, such as his triggers, signs and symptoms of rising anger and methods to reduce angry feelings – which remained an important aspect of his formulation – and which did reduce somewhat after therapy. Furthermore, the good therapeutic relationship that I think we developed allowed him to feel validated and listened-to, which could prove useful for potential psychological interventions in future. Overall, it was difficult to fully address the apparently complex nature of his difficulties within the bounds of the current intervention.

XI.1) RECOMMENDATIONS

Based on the reformulation of Roshan's case, it was recommended that he and his mother undergo a course of Family Therapy, for which they were placed onto the ward's waiting list. However, shortly after the end of the anger intervention, and due to the little time that Roshan spent on the ward in a typical week (i.e. only presenting for anger sessions), his funding was discontinued and he was discharged from the ward. He and his mother were therefore placed instead on the waiting list of local services. This was considered particularly important, given that Roshan intends to remain at home with his mother for the foreseeable future.

XII. REFLECTIONS

As described above, Roshan often digressed in sessions to talk about his mother – and specifically, occasions on which he had become aggressive and violent with her – discussing his often rather extreme violence in a casual, matter-of-fact sort of way. He frequently stated – and it indeed appeared – that he felt no guilt or remorse for such things as dragging his mother across the floor by her hair, throwing her into racks of wine bottles and jumping on her neck as she lay on the floor. The apparently clear (family) systemic factors involved in his anger notwithstanding, it was nevertheless challenging to hear him talk like this, and sometimes a challenge to keep the mitigating influences of his ASD in mind. I spoke to other team members about challenging thoughts and feelings I had, as and when they arose, so as to ventilate and process

them, as I was aware that keeping them to myself could have proved unhelpful. In this way, I was able to develop and maintain a good therapeutic relationship with Roshan. In fact, such was our rapport that, on a few occasions, Roshan told me that I was ‘one of the only ones who understand [what she is like]’. In communicating this, he seemed to be implying that I thought such violent behaviour acceptable, *given his mother’s personality/behaviour*. This made me feel uncomfortable and, at such times, I tried to be clear in reiterating to him my unambiguous condemnation of any violent behaviour, on the one hand, whilst acknowledging that, sometimes, other people can make us feel angry/aggressive, and thereby seeking to avoid invalidating his experience of anger. Overall, in terms of my professional development, I feel that this case has demonstrated to me the potential importance of systemic factors in any given presentation, regardless of the presence or not of apparently fundamental neuropsychological deficits. This will be important, as I intend to pursue a career in Neuropsychology.

REFERENCES

1. Association, A.P., *Diagnostic and Statistical Manual of Mental Disorders: DSM-5 (5th Ed.)*. 2013, Washington, D.C.: American Psychiatric Association.
2. Hill, E.L., *Executive dysfunction in autism*. Trends in cognitive sciences, 2004. **8**(1): p. 26-32.
3. Van Eylen, L., et al., *Executive functioning in autism spectrum disorders: Influence of task and sample characteristics and relation to symptom severity*. European child & adolescent psychiatry, 2015. **24**(11): p. 1399-1417.
4. Brugha, T.S., et al., *Epidemiology of autism spectrum disorders in adults in the community in England*. Arch Gen Psychiatry, 2011. **68**(5): p. 459-65.
5. Lai, M.C., M.V. Lombardo, and S. Baron-Cohen, *Autism*. Lancet, 2014. **383**(9920): p. 896-910.
6. Sofronoff, K., et al., *A randomized controlled trial of a cognitive behavioural intervention for anger management in children diagnosed with Asperger syndrome*. Journal of autism and developmental disorders, 2007. **37**(7): p. 1203-1214.

7. Faraone, S.V., et al., *The worldwide prevalence of ADHD: is it an American condition?* World Psychiatry, 2003. 2(2): p. 104-13.
8. Zametkin, A.J. and M. Ernst, *Problems in the management of attention-deficit-hyperactivity disorder.* N Engl J Med, 1999. 340(1): p. 40-6.
9. Sheppard, B., et al., *ADHD prevalence and association with hoarding behaviors in childhood-onset OCD.* Depress Anxiety, 2010. 27(7): p. 667-74.
10. Barkley, R.A., et al., *The persistence of attention-deficit/hyperactivity disorder into young adulthood as a function of reporting source and definition of disorder.* J Abnorm Psychol, 2002. 111(2): p. 279-89.
11. Lara, C., et al., *Childhood predictors of adult attention-deficit/hyperactivity disorder: results from the World Health Organization World Mental Health Survey Initiative.* Biol Psychiatry, 2009. 65(1): p. 46-54.
12. Ebejer, J.L., et al., *Attention deficit hyperactivity disorder in Australian adults: prevalence, persistence, conduct problems and disadvantage.* PLoS One, 2012. 7(10): p. e47404.
13. Novik, T.S., et al., *Influence of gender on attention-deficit/hyperactivity disorder in Europe--ADORE.* Eur Child Adolesc Psychiatry, 2006. 15 Suppl 1: p. 115-24.
14. Institute, A. *Aetiology of ADHD.* 2016; Available from: <http://www.adhd-institute.com/burden-of-adhd/aetiology/>.
15. Barkley, R.A., *ADHD: Long-term course, adult outcome and comorbid disorders,* in *NIH Consensus Development Conference on Diagnosis and Treatment of Attention Deficit Hyperactivity Disorder.* 1998, National Institutes of Health Continuing Medical Education: Bethesda, Maryland. p. 57-60.
16. Barkley, R.A., et al., *The adolescent outcome of hyperactive children diagnosed by research criteria: I. An 8-year prospective follow-up study.* J Am Acad Child Adolesc Psychiatry, 1990. 29(4): p. 546-57.
17. Biederman, J., et al., *A prospective 4-year follow-up study of attention-deficit hyperactivity and related disorders.* Arch Gen Psychiatry, 1996. 53(5): p. 437-46.

18. Matthies, S. and A. Philipsen, *Comorbidity of Personality Disorders and Adult Attention Deficit Hyperactivity Disorder (ADHD)--Review of Recent Findings*. Curr Psychiatry Rep, 2016. **18**(4): p. 33.
19. Matthies, S.D. and A. Philipsen, *Common ground in Attention Deficit Hyperactivity Disorder (ADHD) and Borderline Personality Disorder (BPD)-review of recent findings*. Borderline Personal Disord Emot Dysregul, 2014. **1**: p. 3.
20. Storebo, O.J. and E. Simonsen, *The Association Between ADHD and Antisocial Personality Disorder (ASPD): A Review*. J Atten Disord, 2016. **20**(10): p. 815-24.
21. Gizer, I.R., C. Ficks, and I.D. Waldman, *Candidate gene studies of ADHD: a meta-analytic review*. Hum Genet, 2009. **126**(1): p. 51-90.
22. Anckarsater, H., et al., *The impact of ADHD and autism spectrum disorders on temperament, character, and personality development*. Am J Psychiatry, 2006. **163**(7): p. 1239-44.
23. Ni, H.C. and S.S. Gau, *Co-occurrence of attention-deficit hyperactivity disorder symptoms with other psychopathology in young adults: parenting style as a moderator*. Compr Psychiatry, 2015. **57**: p. 85-96.
24. Lee, D.O. and O.Y. Ousley, *Attention-deficit hyperactivity disorder symptoms in a clinic sample of children and adolescents with pervasive developmental disorders*. J Child Adolesc Psychopharmacol, 2006. **16**(6): p. 737-46.
25. Gadow, K.D., C.J. DeVincent, and J. Pomeroy, *ADHD symptom subtypes in children with pervasive developmental disorder*. J Autism Dev Disord, 2006. **36**(2): p. 271-83.
26. Mulligan, A., et al., *Autism symptoms in Attention-Deficit/Hyperactivity Disorder: a familial trait which correlates with conduct, oppositional defiant, language and motor disorders*. J Autism Dev Disord, 2009. **39**(2): p. 197-209.
27. Blankenship, K. and N.F. Minshawi, *Behavioral Therapy with an Individual with Asperger's Disorder*. Psychiatry (Edgmont), 2010. **7**(8): p. 38-41.

28. Poppes, P., A.J. van der Putten, and C. Vlaskamp, *Frequency and severity of challenging behaviour in people with profound intellectual and multiple disabilities*. Res Dev Disabil, 2010. **31**(6): p. 1269-75.
29. Castelli, F., et al., *Autism, Asperger syndrome and brain mechanisms for the attribution of mental states to animated shapes*. Brain, 2002. **125**(Pt 8): p. 1839-49.
30. Hill, E., S. Berthoz, and U. Frith, *Brief report: cognitive processing of own emotions in individuals with autistic spectrum disorder and in their relatives*. J Autism Dev Disord, 2004. **34**(2): p. 229-35.
31. Attwood, T., *Cognitive behaviour therapy for children and adults with Asperger's syndrome*. Behaviour Change, 2004. **21**(03): p. 147-161.
32. Ramirez, C.A., et al., *Anger and anger expression in adults with high ADHD symptoms*. Journal of Attention Disorders, 1997. **2**(2): p. 115-128.
33. Richards, T.L., et al., *Driving anger and driving behavior in adults with ADHD*. J Atten Disord, 2006. **10**(1): p. 54-64.
34. Wender, P., *Attention deficit hyperactivity disorder in adults: a wide view of a widespread condition*. Psychiatric Annals, 1997. **27**(8): p. 556-562.
35. Barkley, R.A., *More on the new theory of ADHD*. ADHD Report, 1994. **2**(2): p. 1-4.
36. NICE. *Challenging behaviour and learning disabilities: prevention and interventions for people with learning disabilities whose behaviour challenges*. NICE guideline [NG11]. 2015; Available from: <https://www.nice.org.uk/guidance/ng11?unlid=202191859201610163616>.
37. Kellner, M.H. and J. Tutin, *A school-based anger management program for developmentally and emotionally disabled high school students*. Adolescence, 1995. **30**(120): p. 813-25.
38. Singh, A., et al., *Overview of Attention Deficit Hyperactivity Disorder in Young Children*. Health Psychol Res, 2015. **3**(2): p. 2115.

39. NICE. *Attention deficit hyperactivity disorder: diagnosis and management. Clinical guideline [CG72]*. 2008; Available from: <https://www.nice.org.uk/guidance/cg72?unlid=520684933201611441814>.
40. Novaco, R.W., *Anger control: The development and evaluation of an experimental treatment*. 1975: Lexington.
41. Siegel, J.M., *The multidimensional anger inventory*. Journal of personality and social psychology, 1986. **51**(1): p. 191.
42. Siegel, J.M., *The measurement of anger as a multidimensional construct.*, in *Anger and hostility in cardiovascular and behavioral disorders*, M.A. Chesney and R.H. Rosenman, Editors. 1985, Hemisphere: Washington, D. C. p. 59-82.
43. Siegel, J.M., *The Multidimensional Anger Inventory*, in *Innovations in clinical practice: A source book*, P.A. Keller and S.R. Heyman, Editors. 1987, Professiona Resource Exchange: Sarasota, FL. p. 276-287.
44. Culhane, S.E. and O.F. Morera, *Reliability and validity of the Novaco Anger Scale and provocation inventory (NAS-PI) and state-trait anger expression inventory-2 (STAXI-2) in Hispanic and non-hispanic white student samples*. Hispanic Journal of Behavioral Sciences, 2010. **32**(4): p. 586-606.
45. Wechsler, D., *WAIS-IV: Wechsler adult intelligence scale*. 2008: Pearson San Antonio, TX.
46. Reitan, R.M., *The relation of the trail making test to organic brain damage*. J Consult Psychol, 1955. **19**(5): p. 393-4.
47. Delis, D.C., E. Kaplan, and J.H. Kramer, *Delis-Kaplan Executive Function System (D-KEFS)*. 2001, Pearson Assessment: London.
48. Burgess, P.W. and T. Shallice, *Hayling and Brixton Tests*. 1997, Pearson: London.
49. Association, A.P., *DSM 5*. 2013: American Psychiatric Association.

APPENDIX 1: FULL (ANONYMISED) WAIS-IV^{UK} ASSESSMENT REPORT

Detailed background information regarding Roshan and his presentation can be found in the final assessment report of the [ward], by [Clinical Psychologist] and [Assessment and Referrals Clinician], dated [date]. In summary, and more specifically related to the present assessment report, Roshan was referred to the [ward] in order to address his anger management issues. The present cognitive assessment was therefore undertaken as part of a longitudinal formulation that the Psychology team are constructing regarding Roshan's anger issues – specifically, in order to try to gain a greater understanding of the psychological factors involved in his angry/aggressive responses to potential triggers/provocations (in order to inform further treatment approaches).

Behavioural Observations

Roshan was tested across two sessions at the [ward], with each of these sessions lasting approximately one hour. When I knocked on his door to begin the testing, Roshan was anticipating our appointment and was ready to begin the assessment right away. Prior to the delivery of the testing procedures, Roshan was given a general overview of what the testing would involve and was offered the opportunity to ask questions. He was well-engaged throughout the testing sessions and seemed to be applying his full effort on the tasks. He did not show any obvious signs of fatigue or irritation during testing.

Description of Assessment

Roshan's general intellectual ability was assessed using the UK version of the Wechsler Adult Intelligence Scale (fourth edition; the WAIS-IV^{UK}). The WAIS-IV^{UK} is a standardised, reliable measure of an adult's (i.e. those aged 16-90 years) intellectual functioning comprising of 10 core sub-tests. The WAIS-IV^{UK} groups an individual's score into 4 domains: the Verbal Comprehension Index, which measures verbal abilities; the Perceptual Reasoning Index, an indication of non-verbal ability including manipulation of concrete material; the Working Memory Index, which measures ability to hold and manipulate information in short-term memory; and the Processing Speed Index, which measures cognitive processing efficiency. Scores achieved on each of the sub-scales can be used to calculate a Full Scale Intelligence Quotient (FSIQ). Each of the sub-scales and the FSIQ have a mean score of 100 and a Standard Deviation of 15. Roshan's scores on the WAIS-IV^{UK} were compared to the average scores obtained by a sample of the general population within his age range.

Summary of Performance

Overall Current Level of Intellectual Functioning: the WAIS-IV^{UK}

Verbal Comprehension Index (VCI)

Roshan scored 100, indicating that his ability to process and express verbal material falls into the Average range. He scored at the 50th percentile.

In the Similarities subtest, Roshan was required to identify the way in which two common words, objects or concepts are alike (e.g. 'Horse' and 'Tiger'). Scores are based on the identification of a classification system which is relevant to both words (e.g., 'mammal') or the identification of a specific property common to both words (e.g., both have tails). His score was in the Above Average range¹, which indicates a somewhat elevated level of cognitive flexibility and abstraction. Roshan's Similarities performance was both a statistically- and clinically-significant strength for him, the magnitude of this relatively greater performance being observed in only 2-5% of the general population.

The Vocabulary subtest requires the individual to provide definitions for a selection of words. Points are awarded for good synonyms, a major use and a classification or through describing several features that together encapsulate an understanding of the word. Here, too, Roshan's score fell within the Average range. His Vocabulary performance was in line with the average of his performances in the other VCI subtests.

The Information subtest requires the examinee to answer questions that address a broad range of general knowledge topics, assessing verbal reasoning and conceptualisation. Roshan performed in the Average range on this subtest. Relative to his performances on the other subtests comprising the VCI, Roshan's Information performance was a statistically-significant weakness for him. However, the magnitude of this relatively inferior performance is also observed in

¹ A scaled score from ≤ 3 is described as 'Extremely Low'; a scaled score from 4-5 is described as 'Borderline'; a scaled score from 6-7 is described as 'Low Average'; a scaled score from 8-11 is described as 'Average'; a scaled score from 12-13 is described as 'Above Average'; a scaled score from 14-15 is described as 'Superior'; and a scaled score ≥ 16 is described as 'Very Superior'.

15% of the population, meaning that, though statistically-significant, this difference cannot be considered to be clinically-significant.

Perceptual Reasoning Index (PRI)

ROSHAN scored 88, indicating that his ability to process visual material falls into the Low Average range. He scored at the 21st percentile. There were no statistically- or clinically-significant differences between any of Roshan's performances on the three subtests which comprise the PRI.

In the Block Design subtest, the individual is given blocks which have white sides, red sides and sides that are half-red and half-white. Using these blocks, the examinee has to create designs that match those presented in a booklet as quickly as possible. During this task, Roshan scored in the Low Average range, indicating a broadly normal ability-level, with respect to the perception and recreation of a series of complex designs.

In the Matrix Reasoning subtest, the examinee looks at an incomplete 'matrix' or pattern and is asked to select the missing part of the pattern. It is a measure of visual intelligence, classification and spatial ability, knowledge of part-whole relationships, simultaneous processing and perceptual organisation. On this subtest, Roshan scored in the Average range, indicating that his skills here are in line with those of the general population.

In the Visual Puzzles subtest, working within a specified time-limit, the examinee views a completed puzzle and selects three response options that, when combined, reconstruct the puzzle. Roshan scored in the Average range on this task, as well.

Roshan's VCI score was, statistically, significantly greater than his score for the PRI. However, given that the magnitude of this difference is shared with 19.3% of the general population, it cannot be deemed to be clinically-significant. Nevertheless, it should be noted that his abilities as regards the processing and expression of verbal material, represent a relative strength, compared to his ability to process and analyse visual and spatial information.

It should be noted that, due to an unusually large difference between Roshan's Verbal Comprehension Index score and his Perceptual Reasoning

Index, it was only possible to examine relative strengths and weaknesses for specific subtests within the VCI and PRI and not the following two indices.

Processing Speed Index (PSI)

Roshan scored 102, indicating that his speed of information processing falls within the Average range. He scored at the 55th percentile. There was no difference in Roshan's ability-level on the two subtests that comprise this Index.

Regarding the individual subtests that comprise the PSI, the Symbol Search task requires the examinee to search a group and decide whether the target symbol(s) matches any of the symbols in the search group within a specified time limit. Roshan scored within the Average range on this subtest.

The Coding task requires the individual to copy symbols that are paired with numbers within a specified time limit. Roshan again performed within the Average range on this subtest.

Working Memory Index (WMI)

Roshan scored 97, indicating that his ability to temporarily hold and manipulate information within his short-term memory falls into the Average range. He scored at the 42nd percentile. There was no difference in Roshan's ability-level on the two subtests that comprise this Index.

In the Digit Span subtest, Roshan had to repeat single digit numbers back to me, firstly in order and then reverse them. He could successfully recall 6 digits in order and 5 in reverse order which, overall, placed him in the Average range.

In the Arithmetic subtest, working within a specified time-limit, the individual must mentally solve a series of arithmetic problems. Roshan scored in the Average range on this task, indicating that his ability to briefly hold information in mind whilst at the same manipulating this information is in line with the general population.

Full Scale Intelligence Quotient (FSIQ)

It should be noted that, due to the large difference between Roshan's Verbal Comprehension Index score and his Perceptual Reasoning Index score, his FSIQ could not be validly computed.

SUMMARY: Overall, Roshan's performance and score on the WAIS-IV^{UK} suggests that, although his processing speed is within the average range, relative to others of his age, his verbal comprehension and working memory abilities are slightly lower than the general population, whilst he exhibits a more marked difficulty in his ability to process visual and spatial material. This may mean that Roshan has (and has had) difficulty with everyday skills such as orienting himself in space (e.g., map-reading) and organising things (e.g., clothes, stationery, etc.) in physical space. It may be that his relatively (compared to his poor PRI) better language abilities act to somewhat mask his difficulties with visuo-spatial reasoning.

Table A1: Scores from the WAIS-IV^{UK}

Index/subtest	Score	95% confidence interval	Percentile Rank	Descriptive Category
Verbal Comprehension Index	100	94-106	50	Average
Similarities	13			
Vocabulary	9			
Information	8			
Perceptual Reasoning Index	88	82-95	21	Low Average
Block Design	7			
Matrix Reasoning	8			
Visual Puzzles	9			
Working Memory Index	97	90-104	42	Average
Digit Span	10			
Arithmetic	9			
Processing Speed Index	102	93-110	39	Average
Symbol Search	11			
Coding	10			

Note: Scores related to indices, given in bold, are index scores; scores related to subtests, unbolded, are unscaled total scores.

APPENDIX 2: RAW DATA FROM TESTS OF EXECUTIVE FUNCTIONING

Roshan's performance on selected tests of executive functioning is displayed in Table A2.

Table A2: Roshan's performance on tests of executive functioning

Trail-Making Test		
	Time (seconds)	Percentile
Part A	23	40 th
Errors	0	
Part B	48	40 th -50 th
Errors	0	
Delis-Kaplan Executive Function System (D-KEFS)		
	Raw score	Scaled score
Verbal Fluency		
Letter Fluency	47	13
Category Fluency	36	9
Category Switching	12	8
Switching Accuracy	11	9
The Hayling and Brixton Tests		
	Raw score (s)	Scaled score
Sensible completion	11	5 (Moderate Average)
Unconnected completion	9	6 (Average)
Errors	0	8 (Good)
Total scaled scores	19	6 (Average)
Brixton		
Errors	15	6 (Average)